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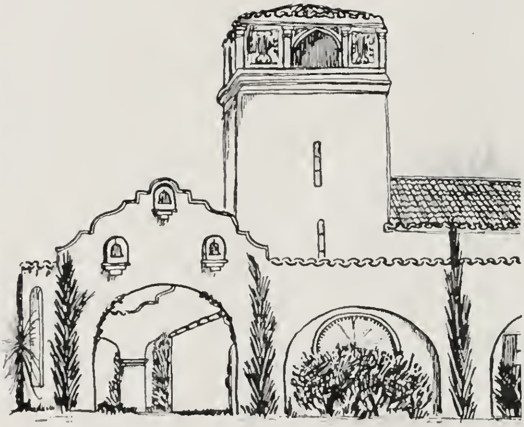

COLLEGE OF OSTEOPATHIC PHYSICIANS
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DISEASES

OF

THE STOMACH

BY

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EDITED, WITH ADDITIONS

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AUTHORIZED TRANSLATION FROM THE GERMAN, UNDER THE
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PREFACE.

THE excellence of the series of monographs issued under the editorship of Professor Nothnagel has been recognized by all who are sufficiently familiar with German to read these works, and the series has found a not inconsiderable proportion of its distribution in this and other English-speaking countries. I have so often heard regret expressed by those whose lack of familiarity with German kept these works beyond their reach, that I was glad of the opportunity to assist in the bringing out of an English edition. It was especially gratifying to find that the prominent specialists who were invited to co-operate by editing separate volumes were as interested as myself in the matter of publication of an English edition. These editors have been requested to make such additions to the original articles as seem necessary to them to bring the articles fully up to date and at the same time to adapt them thoroughly to the American or English reader. The names of the editors alone suffice to assure the profession that in the additions there will be preserved the same high standard of excellence that has been so conspicuous a feature in the original German articles.

In all cases the German author has been consulted with regard to the publication of this edition of his work, and has given specific consent. In one case only it was unfortunately necessary to substitute for the translation of the German article an entirely new one by an American author, on account of a previous arrangement of the German author to issue a translation of his article separately from this series. With this exception the Nothnagel series will be presented intact.

ALFRED STENGEL.

EDITOR'S PREFACE.

The appearance in English of Professor Riegel's book will prove a satisfaction to the many who have awaited its publication, because the words are those of a master who spares neither time nor pains in perfecting his work. With characteristic absence of posing, in simple, strong, and dignified language, the author has presented his subject with such sincerity and clearness that his views will meet almost invariably with ready acceptance. It has been a pleasure to give the work the careful examination necessary, and to compare his statements with those in current literature. The text has required but few modifications and additions to make it thoroughly represent the thought of the year. This is so for the reason that, although many contributions have been published since the appearance of the German edition, some of these have been the thrashing out of old straw, and others have been anticipated by the far-sightedness of the author. To the subjects of periodic vomiting in children, syphilis of the stomach, the etiology of gastric ulcer, the blood in ulcer and cancer, diet in hyperacidity, acute dilatation of the stomach, digestion leukocytosis, benign tumors of the stomach, achylia gastrica, hematemesis of hepatic cirrhosis, congenital stenosis of the pylorus, operative procedure in stomach diseases, and to a few others, some words have been added, but pains have been taken not to change the author's own statements. All new matter presented in the volume is included in brackets, so that the reader will have, as nearly as the translation will admit, the work of Riegel, with the additions of some views for which the editor is alone responsible.

In conclusion, I desire to express my thanks to Professor Alfred Stengel, the editor-in-chief, for thoughtful suggestions, to the publishers for numerous courtesies, and to Dr. Allen A. Jones for assistance in proof-reading and other particulars.

CHARLES G. STOCKTON.

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DISEASES

OF

THE STOMACH

DISEASES OF THE STOMACH.

INTRODUCTORY.

THE present state of our knowledge of diseases of the stomach is radically different from that of twenty years ago, for whereas formerly the pathologic anatomy of gastric lesions was the principal subject of investigation, attention is directed nowadays chiefly to perversions of the physiologic function of the stomach.

No one will deny that the first aim of a clinical diagnosis should be an elucidation of the pathologic anatomy. If we were able to see every gastric ulcer, and to study all the changes of the mucous lining of the organ, this would constitute a great advance. The practising physician, however, is not content with an anatomic diagnosis alone: he must understand the remote factors of the disease, and must know to what extent the different functions of the organ are perverted. If the degree of anatomic change were proportionate to the character and intensity of the perversion of function, all that would be needed would be a diagnosis of the anatomic changes. For the present, however, no such relationship is known to exist, or, if it exists, is not understood. Pathologic anatomy never taught us that in carcinoma the production of hydrochloric acid is reduced, but the clinician discovered this, and later succeeded in explaining it on the basis of the anatomic lesions found. Pathologic anatomy has not taught us that in this or that process hyperacidity must obtain, but clinical investigation has determined this, and even to-day the anatomic basis of this finding is obscure. The physician must direct his treatment toward a reëstablishment of perverted function in all those cases in which he cannot cure the primary disease. In rare cases only will it be possible to institute a radical cure. The treatment advised may be symptomatic, but it is widely different from former symptomatic methods of treatment that were directed exclusively against certain conspicuous symptoms, but not against the fundamental disturbances of physiologic function. Whoever has followed the development of our knowledge of diseases of the stomach will appreciate the refinement of our understanding of perverted function, particularly if he compares it with our crude knowledge of several years ago.

Investigators during the last few years have succeeded in determining the specific disturbances of function that obtain in different diseases of the stomach. This progress was not sudden, and many errors were made and corrected on the way. As in every other line of investigation, the new results were either criticized or overestimated, and it is only within late years that an equilibrium has been established. Nowadays we are not content with determining in a general way in what direction and in what manner the specific functions of the stomach are perverted by different diseases, but we demand that the character and the intensity of functional disturbances be determined in each individual patient. In this manner a complete picture of the disease process may be obtained.

I will endeavor briefly to delineate the history of the views that are prevalent to-day. Kussmaul was the first to recommend the universal employment of the stomach-sound and the stomach-pump (1869), and to demonstrate the great value of these instruments in the treatment of gastric ectasy. I mention Kussmaul's contribution to the study of stomach-diseases first, although it was not originally considered important from a diagnostic point of view, and was primarily a method of treatment. The introduction of the sound for therapeutic purposes, however, taught us to utilize the gastric contents that were washed out in this procedure for diagnostic purposes. It is surprising that this was not done at that time.

Leube (1871) deserves the credit of having employed the stomach-tube for diagnostic purposes. This investigator attempted to determine two points—first, the time of digestion; second, the degree of gastric secretion. Leube's method of examining the duration of digestion is universally employed to-day, notwithstanding the introduction of many other methods. His method, on the other hand, of determining the degree of secretion has not been adopted, because it does not give uniform results when instituted in cases where the stomach is empty. It is probably due to this unsatisfactory result that Leube's proposition to utilize the tube for diagnostic purposes was not adopted at once.

In 1879 a dissertation by von der Velden appeared from Kussmaul's Clinic that revived this subject. The author analyzed the stomach-contents of a series of patients with ectasy, and found that in a number of the cases the stomach gave certain hydrochloric acid reactions with stains that he employed, and in others did not. He determined that in those ectasies that were due to carcinoma no hydrochloric reaction was given, whereas in those that were not due to carcinoma the reaction was positive. Von der Velden concluded from the absence of this color-reaction in carcinomatous ectasies that no hydrochloric acid was secreted in this condition. His report remained altogether unnoticed for a long time, and where it was noticed, was violently combated (Ewald). Very soon after the appearance of von der Velden's report I repeated his experiments and corroborated his results. I found, however, that the addition even of large quantities of hydrochloric acid to stomach-contents that gave no reaction did not cause the appearance of hydrochloric acid reactions. I deduced from these experiments that the hydrochloric acid that was noticed was either neutralized or entered into some combination.

I did not limit my experiments to the analysis of stomach-contents in ectasies, but extended them to the analysis of stomach-contents in other obstinate diseases of the stomach. In order to obtain values that could be compared it appeared necessary to administer a test-meal of uniform composition to these patients. I, therefore, gave a test-meal that was calculated to reveal the functional powers of the stomach from a chemical point of view, following in this respect Leube's method for the elucidation of the motor function.

I did not, however, follow Leube's method of examining the secretion of the stomach when the organ was empty, but attempted to make my tests with stomach-contents that was pumped out at the height of digestion and after the introduction of one of my test-meals. The material removed was examined for its color-reactions and for its digestive powers.

Jaworski, Boas and Ewald, Klemperer, Sée, and others later on recommended different kinds of test-meals. My test-meal and Ewald-Boas' test-breakfast are the ones that are in use to-day.

In continuing and extending von der Velden's experiments we found very soon that the absence of color-reactions was not specific for carcinoma of the pylorus. One year after the appearance of von der Velden's report Edinger (1880) examined a number of cases of amyloid degeneration of the gastric mucosa in my clinic, and found that here, too, the color-reaction was negative—that is, that no free hydrochloric acid was present. This did not impair the significance of von der Velden's observations, but it demonstrated for the first time that the absence of free hydrochloric acid was not absolutely pathognomonic for carcinoma of the stomach. I was enabled to demonstrate some years afterward that cauterization of the stomach-wall could cause free hydrochloric acid to disappear.

During the next few years a large number of case-reports were published, some of them corroborative, some of them contradictory. Many investigators attempted to discover new color-reagents for hydrochloric acid in the stomach-contents. We will only mention the coloring-matter of red wine, rosolic acid, benzopurpurin, raspberry stain, smaragd green, malachite green, etc. All these have been relegated to oblivion.

Some years later Congo-red (1886) and phloroglucin vanillin (1887) were introduced. These are still in use. Only one of the many reagents that were discovered before this time is still employed—namely, Uffelmann's reagent (a mixture of chlorid of iron and carbolic acid as a test for lactic acid).

This question of hydrochloric acid that von der Velden first introduced into medical science soon commanded the interest of clinicians. In 1886, however, an investigation by Cahn and von Mering appeared that threatened to reverse all the decisions arrived at so far. These authors succeeded by a special method in discovering hydrochloric acid in stomach-contents, even when none of the color-reactions was positive.

Because of this discovery they denied the validity of all the color-reactions, notwithstanding the statements made by von der Velden and myself, and claimed that far from being absent, hydrochloric acid was always present in carcinomatous stomach-contents. I induced my assistants, Honigmann and von Noorden, to repeat Cahn-von Mering's experiments. I will describe these investigations at some length in this place, because the results obtained threw a great deal of light on this hydrochloric acid question. Assuming even that the results obtained by Cahn and von Mering were correct, the question was not settled by any means. A large number of experiments have taught me that the principal difference between the stomach-contents of carcinomatous and non-carcinomatous cases was not alone the presence or the absence of a positive color-reaction. Another, and possibly more important, difference consists in the power of the one to digest a disc of albumin, whereas the other does not possess this power; in other words, the stomach-contents of carcinomatous cases had lost their digestive power, whereas the stomach-contents of non-carcinomatous cases still retained it. If hydrochloric acid was added to the stomach-contents of cases of gastric carcinoma in such quantities that a distinct color-reaction would ordinarily be obtained, and sufficient ordinarily to digest a disc of albumin in the presence of sufficient pepsin, this stomach-contents neither gave hydrochloric acid reactions nor was it able to dissolve the disc of albumin. This demonstrated conclusively that the hydrochloric acid had become inactive, had entered into some combination, or had been used up.

Honigmann and von Noorden were asked to investigate this peculiarity. In the experiments that were made, according to Cahn and von Mering, my assistants could determine that all stomach-contents that did not give a color-reaction still contained an acid residue that colored litmus red, but did not give specific reactions and could not digest albumin. If they added a definite quantity of hydrochloric acid of known titration to such gastric contents, they did not find the quantity added after a time, but always determined a deficit of hydrochloric acid. This demonstrated that the acid residue described by Cahn-von Mering could not be free hydrochloric acid. If this had been the case, the free hydrochloric acid and the amount added should have increased the amount of hydrochloric acid found in the mixture. It was also shown that the amount of hydrochloric acid present was too small for the hydrochloric affinities of the stomach-contents, and in order to establish normal proportions it was necessary in these cases to add a larger amount of hydrochloric acid. Honigmann and von Noorden determined this amount by estimating the amount of hydrochloric acid necessary to bring about a distinct hydrochloric acid reaction in the stomach-contents. As soon as the reaction appeared it was known that all the hydrochloric acid affinities were saturated and that free acid was present. These examinations not only explained the absence of this reaction in carcinoma, but, to quote from Martius, attached a new significance to the presence of free hydrochloric acid. A stomach-contents that contains hydrochloric acid is capable of fulfiling its function if it contains free, or, as we often say, excessive, hydrochloric acid. On the other hand, if free hydrochloric acid is absent and the color-reactions are negative, the amount of digestive secretion is insufficient.

These experiments reëstablished the significance of color-reactions for free acid, or rather for free hydrochloric acid. For a long time clinicians were satisfied with these simple color-reagents, as they seemed to answer the question that was of paramount importance to the practising physician—viz., whether the stomach secreted a sufficient quantity of gastric juice or not.

Gradually, however, the demand was formulated to obtain a numerical expression of the amount of hydrochloric acid secreted. Careful investigations demonstrated that even in those cases where the color-reactions and digestion tests were positive, the intensity of these reactions fluctuated within wide boundaries. In this manner the demand for a quantitative determination of the acid, particularly in those cases where color-reaction was positive, was created. The total acidity was determined, and three possibilities were recognized: chemism was normal, or there was superacidity or subacidity. For some time these methods sufficed; gradually, however, new doubts arose. The methods were considered satisfactory if the color-reaction was positive; if they were negative, the methods were regarded as insufficient, for it was argued that even though color-reaction was negative, a great deal of hydrochloric acid might be present in combination. A method had to be found, therefore, to determine the total quantity of hydrochloric acid secreted—above all, of that which was in combination. For a number of years nearly all investigators in this field have directed their efforts toward the discovery of exact methods for this quantitative determination. The majority of these methods are deficient because they do not differentiate distinctly between total hydrochloric acid, free, and combined hydrochloric acid, so that much confusion has arisen.

It would lead us too far to discuss the numerous methods that have been worked out in the course of time. I must refer to the paragraph on methods of hydrochloric acid determination. If any one of my readers is particularly interested in this question and its development, I refer him to the exhaustive publication by Martius and Lüttke.[1] Here they will find an exhaustive criticism of all the older methods. I will only mention in this place that no absolutely correct method has so far been discovered.

If we ask what the result of all these endeavors has been, we must acknowledge that the results obtained are not proportionate to the amount of work done. Neither diagnosis nor therapy has profited very much from all these investigations. It is interesting, of course, to determine the total quantity, the combined, and the free hydrochloric acid in every case of stomach-disease. It is, above all, interesting to determine the quantity of combined hydrochloric acid in cases of subacidity, but nothing practical is gained by these determinations. If a stomach-contents contains no free hydrochloric acid and is not able to digest a disc of albumin, then there is subacidity, and the secretion of gastric juice is insufficient. If in a case of this character we determine the amount of hydrochloric acid, we obtain a certain figure for this amount, but practically it is of no value to us. We may determine that one case has more, the other less, combined hydrochloric acid, but this does not help us in either the diagnosis or the treatment of the case.

---

[1] *The Gastric Juice of Man*, 1892.

The method of Honigmann and von Noorden that we have described above might be of more value—namely, the addition of titrated hydrochloric acid until free hydrochloric acid appears, and indicates a deficit. The knowledge of this deficit will certainly give us a clearer conception of the degree of the acid insufficiency than the analytic determination of the amount of hydrochloric acid present. The practising physician can usually get along very well without any of these methods, and if he employs any at all, he will probably find that the old inductive ones that were recommended some ten years ago are sufficient for his purposes.

From what we have said, the impression might be gained that the most important feature of the pathology of stomach-diseases is this hydrochloric acid question. As a matter of fact, this question dominated the field for a long time, and it is due to this that some of the criticism of the modern trend of gastric pathology seems really to be justified. Personally, I have always maintained a conservative attitude toward these frantic endeavors to determine minute traces of hydrochloric acid, and have always emphasized that the chemical examination of the stomach-contents is valuable only in combination with other findings.

As in the case of kidney diseases, the exact determination of albumin is not sufficient, and as in the case of this organ the quantity of the urine, its specific gravity, the quantity and the character of the formed elements, etc., are all of importance, so we must look for a *combination* of factors in the interpretation of diseases of the stomach.

If we want to understand the character of the disease, we must examine the stomach and its functions in all directions. It is not sufficient to know how much hydrochloric acid is excreted, but we must know what the stomach can do with meat, with carbohydrates, what its motor powers and what its absorbing capacity are. For these reasons I have emphasized for years that it is not sufficient to determine the hydrochloric acid after a test-meal, but that it is just as important to study the quantity, the appearance, the color, and the physical constitution of the gastric contents, as well as the formation of gas and other factors, and that only in this way can a correct estimate of the total functional powers of the stomach be obtained.

As a matter of fact, the hydrochloric acid question no longer occupies the front rank in the pathology of stomach-diseases, and of late clinicians have endeavored to extend our knowledge of the different diseases of the stomach in other directions. Particular attention has been given to disturbances of motility, and their significance in the disturbances of secretion is generally recognized. Impairment of the absorptive powers has been studied, and the significance of gas-formation or of fermentative processes that are accompanied with the development of gas has been emphasized. As a result, the view that I expressed many years ago is now generally accepted—namely, that however interesting or important the disturbances of the secretion of gastric juice may be, they are important only in connection with the results of all methods of examination. In heart-disease auscultation is valuable, but not sufficient to make a diagnosis, and in stomach-diseases a determination of the hydrochloric acid alone is not sufficient. Only in combination with the subjective symptoms of the patient, the determination of the size and position of the stomach, its motor and absorbing powers, is the determination of hydrochloric acid of value.

The chief aim of the physician should be to heal and to help. The practising physician is justified in gauging progress in any one field of the practice of medicine from the advance that has been made in the treatment of disease. If we apply this axiom to the group of diseases under discussion, we can see that within the last twenty years real progress has been made. It is true much remains to be done, but we can see to-day that with a better understanding of the nature of stomach-diseases better treatment has been inaugurated.

We are enabled to-day to relieve, and in some cases to cure, a large number of diseases that baffled all attempts at treatment a few years ago, because we did not understand their nature at that time. Patients that were given up are cured. In part we owe this progress to surgery, and we may expect that medicine and surgery combined will lead to further practical results in this field.

METHODS OF EXAMINATION IN DISEASES OF THE STOMACH.

The Anamnesis.—As in all other diseases, a record of the present state of the disease should be made before the history is elicited. It may seem superfluous to speak at all about the significance of the history and the method of eliciting it in diseases of the stomach. Things are, however, a little peculiar in the case of this organ. In the majority of other diseases it is usually sufficient to obtain a history of the patient in his own words, and to have him report his symptoms and the origin of his troubles. Every clinician who has had much to do with stomach-cases knows that in this category the statements of the patient are usually altogether insufficient, and have to be supplemented by answers to questions formulated directly by the examining physician.

In order to determine the important points in the history of these cases the clinician should be thoroughly familiar with the different disease-pictures; in many cases one who has this experience will be induced to make particular inquiry in regard to some one symptom that the patient describes, but that he attaches very little importance to.

It is impossible to formulate a general scheme for the anamnesis of these cases. Certain practical features, however, may be emphasized that apply to every case. In the same manner as the examination of the case, the questions in regard to the history should be asked in a definite and regular way. If this is not done, there is always danger of overlooking some important point.

The anamnesis will not be confined to the trouble that the patient complains of at the time, but will attempt to trace the disease to its origin, to elicit its causes, and its mode of onset.

The history should include the following points:

(*a*) Local subjective disturbances, pressure, feeling of fulness, pain, restlessness, etc., also the beginning and the probable cause and development of these symptoms.

(*b*) Nausea, belching [regurgitation], vomiting, vomiting of blood.

(*c*) The appetite and the thirst.

(*d*) The stools.

(*e*) The general health, the strength of the patient, and the hereditary features.

It is not enough to ask the patient general questions in regard to pain or appetite, and the inquiry should be varied according to each disease and the peculiarities of each individual case. It would lead us too far to discuss the different points—for instance, in regard to the calling and mode of life, the diet, etc.; we will discuss them later, in the sections on the different diseases. In this place we will only discuss a few general points of view:

(*a*) *The Local Subjective Symptoms.*—Disturbances of deglutition are important. Pain should be definitely defined as to time, regularity or irregularity, paroxysmal character, localization, radiation, character, intensity, duration, the influence of taking food upon it, particularly the influence of certain articles of food; all these points are important and vary in different diseases.

It is also important to establish whether the pain is present at all times or only when the patient occupies certain positions. Some diseases of the stomach are not painful at all; in others, pain is the rule; in ulcer, pain appears immediately after eating; in other diseases much later. The kind of diet frequently exercises an influence on the causation of the pain, and in some forms of diseases, as in continuous secretion of gastric juice, there is a nocturnal exacerbation of the pain, occurring at a time when no ingesta are present within the stomach.

Pain may be diffuse, or strictly limited to some small circumscribed area. Sometimes pain disappears when food is taken; at others it is caused by eating; in other cases again the sensation complained of is not really pain, but a feeling of pressure and fulness in the gastric region. The time, duration, and the coincidence with certain digestive periods should be elicited in the case of these sensations.

(*b*) *Belching, Nausea [Regurgitation], Vomiting, Vomiting of Blood.*—Belching may be absent, or may be very violent and continuous. It is well to determine whether it occurs while the stomach is full or empty, whether the gas is tasteless and odorless or acid, or has some other characteristics. Fetid belching is a symptom of fermentative and putrefactive processes in the stomach. If belching is due to some other nervous disorder, the gas is usually tasteless and odorless, but the condition is an obstinate one. In some cases belching occurs only after the ingestion of certain articles of food; in other cases it is continuous. Attention should always be paid to heartburn—so-called pyrosis.

Nausea and vomiting are seen in a large number of diseases of the stomach. There are, however, great differences in this respect that are diagnostically of significance. The time of vomiting is important—that is, whether it occurs when the stomach is full or empty or in some other period of digestion. In many cases nausea and vomiting occur every day at a regular time; in others, vomiting is irregular and occurs at intervals. Some patients state that the vomit is acid, acrid, and burning; in others the taste is that of the food. The quantity and the microscopic appearance of the vomit are particularly important; sometimes it consists of a large quantity of a cloudy fluid mixed with fine particles of amylaceous material; in others, the food is hardly changed by digestion. Sometimes there is an abundant admixture of mucus or of bile. Frequently the patients state that particles of food are vomited that were eaten several days before. It is frequently important to determine whether or not the patients feel relieved after vomiting.

In many diseases, as in certain nervous troubles, nausea appears when the stomach is empty; in other diseases it follows the ingestion of food; in still others it occurs several hours after eating. Most cases of vomiting are preceded by a feeling of nausea. There are, however, certain forms of vomiting in which nausea is never experienced.

It is particularly important to determine whether or not blood has been vomited. It is necessary to be very careful in accepting statements of the patient himself in this respect; in fact, we can never be certain that the blood was vomited until we have a careful description of the appearance of the vomit.

[Regurgitation differs from belching and also from vomiting. The term applies to the act of bringing up the stomach-contents mouthful by mouthful, without the complicated mechanism of vomiting, and usually without nausea. The cardia relaxes, the intragastric pressure is increased, the chyme is propelled into the pharynx or mouth. The symptom may arise from stenosis at the cardia, in which case the regurgitation is from the distended esophagus rather than from the stomach. More often regurgitation is from the stomach. It is usually associated with a functional gastric disorder, but it sometimes occurs in structural disease of the stomach. When the gastric digestion is sufficiently advanced and the stomach-contents has become acid as a result of normal secretion, the regurgitated fluid produces a sour, burning sensation in the esophagus and pharynx, and is usually described as heartburn or pyrosis. When the regurgitated fluid is bitter, the taste usually depends upon the presence of peptone, and when depending upon the acids of fermentation, it may be especially disagreeable. On the other hand, the stomach-contents may be regurgitated as it was taken. This may result from the early appearance of the symptom before digestion has normally begun, or later, when the digestion has been delayed. This symptom is sometimes the expression of hysteria; and at other times it results from motor irritability of the stomach and relaxation of the cardia, the result of a neurasthenic state.—Ed.]

(c) *The Appetite and the Thirst.*—The appetite is naturally very important. The statements of the patient are usually very unreliable in this respect. The statement that there is loss of appetite should never be accepted, but the exact mode of life of the patient should be elicited, and a careful list of the articles of food that he has been in the habit of eating should be obtained. Only in this manner will it be possible to determine whether the patient is eating enough, what articles of food he avoids, and what articles he favors.

The disturbances of the appetite vary greatly according to the quantity and the quality of the food. There are cases of complete anorexia or of exaggerated hunger—bulimia. Between the two, numerous stages are seen.

Many patients claim to have lost their appetite. If careful inquiry is instituted, it will be found that they do not eat because they suffer from pain after eating. If the pain can be removed, their appetite usually returns.

In certain forms of stomach-disease the appetite is really reduced. This is particularly the case in severe forms of catarrh and in carcinoma. Patients with carcinoma of the stomach frequently have a positive disgust for meat. On the other hand, carcinomatous cases are occasionally seen that retain their appetite for a long time, even after the peptic powers of the stomach have been considerably reduced. According to my experience, this is seen particularly in carcinomata of the body of the stomach, provided there is no motor insufficiency. It is less frequently seen in carcinomatous stenosis of the pylorus. Notwithstanding the fact that the secretion of gastric juice is greatly reduced, the stomach is frequently found to be empty four hours after a test-meal, and the patient may enjoy a good appetite, and, if the diet is correctly selected, maintain his weight.

In some forms of stomach-disease, as in hyperacidity and hypersecretion, the appetite is frequently increased. In certain diseases of the stomach the appetite varies greatly.

Thirst also varies. In ectasy of the stomach of high degree following stenosis of the pylorus, or in the atonic forms of ectasy, thirst is frequently exaggerated. The same symptoms may be observed in many neuroses of the stomach.

(d) *The Stools.*—Many patients suffer from disordered stools; constipation is more frequent than diarrhea. In many cases the two conditions alternate. It is important to observe and to regulate the stools before any deductions in regard to the nature of the stomach-disease are attempted, from the sluggish or active character of the intestinal functions.

It is not well to rely on statements made by the patient in regard to the character of the stools. They should always be controlled.

The physician should determine by personal observation whether mucus, blood, pus, or other abnormal constituents are present in the stools. It is also necessary, of course, to find out what laxatives the patient may have used. If blood is found in the stools, a careful rectal examination should always be made. [A similar course should be followed in case of diarrhea or constipation.—Ed.]

(*e*) *The General Health and the Strength of the Patient.*—The general health and nutrition of the patient are, of course, important in judging of the nature of the disease. If there is great emaciation within a short time, some serious disease of the stomach, particularly carcinoma, should be suspected, even though no tumor is palpable. On the other hand, if the patient has been a sufferer from stomach symptoms for many years, carcinoma should never be diagnosed, even though the organ is found to be ectatic and a tumor can be felt, for in a case of this kind the tumor can only be secondary to some other disease of the stomach that has existed for a long time, or it is not carcinoma. I have a patient in whom ectasy of high degree and a tumor of the pyloric region were diagnosed some ten years ago. This lady is well nourished to-day, so that her case cannot possibly be one of carcinoma.

The diet and the general treatment of the patient preceding the examination are always important. In clinics and hospitals we see how much can be accomplished by an appropriate diet and correct treatment. In private practice and in polyclinic work it is much more difficult to procure the correct diet and enforce all necessary measures. It is frequently possible in clinical work to increase the weight of the patient within a short time, even after failure in this respect in private practice. For this reason statements by the patients that they do not look well, or that they are losing flesh, does not necessarily imply that they are afflicted with some severe and incurable disease of the stomach. The duration of the disease, the mode of life, and the treatment up to date will all have to be considered.

Heredity in general is not important. Even in carcinoma of the stomach only a small proportion of cases give a history of a similar disease among their ancestors. The hereditary transmission of a neuropathic taint is frequently significant and important.

So much about the anamnesis. In this place we could only call attention to some of the principal points. We refer to the sections on the individual diseases for all the details.

PHYSICAL METHODS OF EXAMINATION.

After the history of the case has been obtained, the patient should be submitted to an objective examination. In describing the objective, particularly the physical, methods of examination, I will limit myself, of course, to those that apply to the stomach. No intelligent physician, however, will commit the fundamental error of limiting his examination to the stomach, but will include all the other organs. Even though the statements of the patient point almost positively to some disease involving the stomach alone, it is still necessary to examine all the organs of the chest and the abdomen, to analyze the urine, etc. In surgical diseases it is often possible to limit the examination to the diseased organ; in internal medicine this is not practical, and an examination of all organs is absolutely necessary before the physician can thoroughly understand the disease. In practice we frequently see physicians limiting their examination to the one organ that the patient designates. We may say that no organ can be so misleading in this respect as the stomach, and many other diseases are hidden behind the semblance of some stomach-disease. Secondary disturbances of the functions of the stomach are seen in a great variety of diseases, and the patients are very liable to complain of these gastric symptoms alone because they distress them most.

The physical examination of the stomach includes methods that are employed in the case of all the other organs, and some that are peculiar to the stomach. The first include inspection, palpation, percussion, and auscultation—all methods that have been carried out for a long time, and that of late years have, unfortunately, been relegated to the background, underestimated, and discarded in favor of chemical methods of examination. The methods peculiar to the stomach are artificial inflation, artificial transillumination and illumination of the stomach, and the employment of the stomach-tube.

Inspection.—Inspection, as in the case of the thoracic and abdominal organs in general, plays an important rôle in diseases of the stomach. The examination of the heart, for instance, should always be begun with an inspection of the precordial region, and in the case of this organ it would be a fundamental error to begin with auscultation before having determined by inspection, palpation, and percussion the position and the character of the apex-beat, the configuration of the cardiac region, the size and position of the heart, etc. The same applies to the stomach, and the examination should never be begun with palpation. There should be a system in all examinations of the body and of single organs, and only that clinician who adopts this systematic method will be certain not to overlook this or that symptom. This may seem self-evident, but my experience has taught me that it needs to be particularly emphasized in the case of the organ we are discussing.

Before beginning the examination of the stomach, the general nutrition, the complexion, and the features of the patient should be noted. The state of the general nutrition is very important, and frequently gives us valuable clues. If the patient is very much emaciated, the significance of this finding will be different according to the duration of the disease; if the patient has been emaciated for many years and has been a sufferer from some stomach-trouble that was complicated by frequent vomiting, this symptom will have a different signification than it would if the patient had emaciated rapidly within the course of a few months.

Painful diseases of the stomach, particularly if they are of long duration, frequently produce a peculiar suffering expression of the face. Carcinoma cases often have a peculiar ashy complexion. This coloration of the skin is, however, not pathognomonic, for we frequently see cases of carcinoma, particularly among young people, whose appearance belies the existence of so severe a disease. On the other hand, we frequently see cases of ectasy of long duration that are not due to carcinoma, who are extremely emaciated, and have the peculiar ashy color that is not distinguishable from that of cases of carcinoma. The general appearance and color of the face, the facial expression, and the degree of emaciation are certainly all important points. If, for instance, a patient suddenly turns pale, the possibility of hemorrhage must be considered, particularly if the general nutrition is well maintained and the stomach-disease is a painful one. I have succeeded in a number of ulcer cases in diagnosing hemorrhage from the sudden appearance of pallor, even though no blood was vomited. The subsequent examination of the stools would corroborate the correctness of this view.

An examination of the digestive organs should begin with the inspection of the buccal cavity. The teeth, gums, tongue, palate, and pharynx should be examined. If the teeth are carious, or if many teeth are absent, it is impossible for the patient to masticate and insalivate his food, so that here may be a cause for digestive disturbance.

Sticker[1] and Biernacki[2] have studied the relation of buccal digestion to general digestion in my clinic. If the test-breakfast was introduced into the stomach through a tube and the admixture of saliva avoided, the motor and secretory powers of the stomach seemed reduced. It would lead us too far to analyze this question in detail; we will only mention that Biernacki has shown that the passage of food through the mouth is significant in the sense that it imparts a suitable reaction to the bolus of food, for stomach-digestion seems to be most active if the food that enters it is neutral or slightly acid. A certain diastatic action is, of course, also carried on in the mouth. The experiments of Biernacki show that a thorough digestion is more or less dependent on the normal character of all the structures of the mouth. It is for this reason chiefly that in each stomach-case a careful inspection of the mouth should be made.

In old people one of the most prolific causes of dyspepsia is the lack of teeth. The same applies to artificial teeth that do not fit well and that irritate the mucous lining of the mouth. The teeth should be well taken care of, and artificial teeth should be frequently cleaned in order to prevent the development of fungi in the mouth. The latter may seriously interfere with gastric digestion.

The next organ to examine is the tongue. Older physicians were wont to attach a great deal of importance to the appearance of the tongue in diseases of the stomach. The tongue was for them, so to say, the mirror of the stomach; nowadays physicians no longer hold these views. There is a divergence of opinion in regard to lingual symptoms in the diagnosis of diseases of the stomach. Boas[1] claims that no diagnostic significance can be attached to the appearance of the tongue, or at best a very slight one. He finds that a coated tongue may be seen in cases that enjoy a splendid appetite and an excellent digestion, and that, on the other hand, patients are found who suffer for many weeks or months from anorexia, but whose tongue is perfectly clean. According to Boas, the appearance of the tongue is nothing more nor less than an index or an impression of the condition of the buccal cavity. In all cases where the tongue is bad, caries, periodontitis [alveolaris purulenta], gingivitis, stomatitis, pharyngitis, salivation, and perversion of the secretion of saliva, etc., will be found.

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[1] Sticker, *Volkmann's Samml. klin. Vorträge*, 1887, No. 297.

[2] Biernacki, *Zeitschr. f. klin. Med.*, vol. xxi.

Bouveret[2] occupies a different stand. He believes that gastric disturbances exercise a certain influence on the appearance of the tongue. He claims to have seen many people with a clean tongue whose digestion was good, but whose teeth were bad, and, on the other hand, he claims never to have seen a case of catarrh of the stomach, of nervous dyspepsia, whose tongue was not more or less coated.

From my own experience, I should say that the significance of the tongue in the diagnosis of diseases of the stomach is very subordinate. Sufferers from diseases of the stomach are frequently seen whose tongues appear perfectly normal, and, inversely, many people with healthy stomachs are afflicted with a coated tongue. The tongue, therefore, is in no manner a mirror of the mucous lining of the stomach. I have found, however, that those diseases of the stomach that are characterized by an excessive secretion of hydrochloric acid are more apt to show clean red tongues than those in which the secretion of gastric juice is decreased. On the other hand, it is certain that the tongue may look well, and the stomach at the same time be very much diseased. The relation between the appetite and the appearance of the tongue is slightly different, for the tongue may appear normal and the appetite be decreased, or, inversely, the tongue may be coated and the appetite be relatively good. In general, however, loss of appetite is usually accompanied by a coated tongue.

We say, therefore, that the appearance of the tongue does not permit us to draw any definite conclusions in regard to the character and the significance of diseases of the stomach.

It is also necessary to study the appearance of the soft palate, the tonsils, and the pharynx. Chronic inflammation of the pharynx is principally seen in patients who are smokers and drinkers. Chronic pharyngitis may simulate some disease of the stomach. The appetite is frequently decreased, and there may be reflex vomiting, nausea, and similar symptoms. Appropriate treatment of this pharyngitis will often cause the stomach symptoms to disappear. In every case, therefore, of dyspeptic disorders a careful inspection of the pharynx should be made.

Inspection of the abdomen may, under certain conditions and in certain diseases of the stomach, give valuable clues. In most diseases of the stomach inspection of the gastric region furnishes very little information. The patient should be placed so that the abdominal walls are relaxed, and should be instructed to lie flat on his back and to breathe slowly.

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[1] *Diagnostik und Therapie der Magenkrankheiten*, third edition.

[2] *Traité des mal. de l'estomac*, Paris, 1893.

If the patient is obese, this renders the examination more difficult. Occasionally inspection alone will determine the size, the position, and the outline of the stomach. In individuals who are very much emaciated, and in whom the stomach is ectatic and situated low down, the outline of the stomach may occasionally be seen through the abdominal walls. The region of the stomach appears to protrude above the level of the abdomen, and the course of the greater and the lesser curvature is clearly distinguishable. In some cases a tumor may be seen in the region of the pylorus that prominently elevates the abdominal walls and that will be observed to belong to the stomach. In cases of this kind, which, of course, are rare, the diagnosis of ectasy can frequently be made, and if the appearance of the patient, the history, and the course of the disease coincide, carcinoma may be suspected. Another symptom that points in this direction is a very conspicuous peristaltic motion of the portion of the dilated stomach that protrudes. If this symptom is observed, the diagnosis of cancer becomes still more probable. As a rule, however, peristaltic motion of the stomach is not seen.

In some cases, however, we see a very active peristaltic motion in the region of the stomach (the so-called peristaltic unrest of Kussmaul[1]). The organ is quiet only when it is empty, and not always then. This peristaltic unrest is seen particularly in cases where the stomach is very much dilated, where the musculature is hypertrophic, and in which stenosis of the pylorus or of the duodenum exists. In these cases there is hypertrophy of the muscularis of the stomach as a result of the increased resistance, and excessive peristalsis is due to the exaggerated muscular action of the stomach-walls.

Kussmaul has attempted in these cases of excessive peristaltic unrest to measure the time occupied by the peristaltic waves in passing from the linea alba below the umbilicus in an upward direction and to the right to the lower margin of the liver. According to my personal observations, this time may vary.

At times different, broad waves of unequal amplitude are seen to move in a curved direction from left to right along the lower half of the abdomen. This movement is sometimes seen as soon as the abdomen is denuded, and appears with particular distinctness if the region of the stomach is kneaded or tapped or if the patient is given a Seidlitz powder.

As a rule, dilatation of the stomach causes the organ to move downward and to occupy a more nearly vertical position. The latter change of position is sometimes seen without ectasy, particularly in women. This condition cannot, as a rule, be discovered on simple inspection if it is uncomplicated; it may be the result of arrested development, or it may be acquired as a result of pressure exercised by lacing. The liver is at the same time moved downward and inward, so that the movable pylorus is forced downward and to the left; this causes the pyloric portion of the stomach, as a whole, to move downward and toward the left side of the abdomen below the navel, so that the lowest portion of the stomach, in case the organ is vertical, may be found below the navel, although the dimensions of the stomach remain normal.

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[1] "Die peristaltische Unruhe des Magens," *Volkmann's Samml. klin. Vorträge*, 1880, No. 181.

Occasionally the rare form of hour-glass contraction of the stomach can be discovered by inspection of the abdomen. All these abnormalities of form and position can be more readily recognized if the stomach is inflated with carbonic acid gas, air, or water.

Peristaltic unrest is most frequently seen in ectasies that are due to some mechanical obstruction in the pyloric portion of the stomach or in its vicinity. Kussmaul has called attention to the fact that it is most pronounced in those cases of dilatation that are accompanied by an increased formation of acid,—hyperacidity,—and particularly hypersecretion; it is less frequent and less pronounced in carcinomatous forms of pyloric stenosis. There are still other cases, as Kussmaul says, that are neurotic.

Tumors of the stomach rarely grow so large as to be discoverable by inspection. If they are visible, it is often possible to determine whether they possess peristaltic motility or not. Changes in the position of the growth when the stomach is full or empty can also be seen; sometimes they remain stationary, immobile, whether the stomach is full or empty; in others they move.

In some instances the region of the stomach will be retracted, particularly in stenosis of the cardia. Retraction of the epigastrium may sometimes be seen in ectasies of the stomach of high degree, particularly if the organ is moved downward and there is gastroptosis, and the organ occupies a vertical position. In both instances the lower boundary of the stomach may reach as far downward as the symphysis.

All the changes enumerated are still more distinct if the stomach is artificially inflated. We will speak of this later.

Dilatation of the colon can occasionally be seen in very emaciated cases with relaxed abdominal walls; the dilated colon may appear as two distinct ridges on either side of the abdomen.

[Inspection may be facilitated and the success of the method greatly enhanced by placing the patient upon a raised table, the head toward a window, the shades of which are so arranged that the light enters on a plane only slightly above that of the patient—the rays of light directed from the head toward the feet. Standing now toward the foot of the table and bending from side to side, the examiner is able, by a little experience, to make out delicate shadows cast by the slight inequalities of the abdomen. These shadows are seen to move with respiration. By resorting to this method, the size, shape, and position of the stomach can often be made out when ordinary methods of inspection are unsatisfactory. This manœuver is especially valuable in discovering the gastric and intestinal peristalsis, the presence of enlargements, and, to some extent, the relations of the abdominal viscera. This method I have long practised, and can testify to its utility.

Knapp[1] has recently suggested this modification: with the patient placed in the position described, the observer stands at the side or at the shoulders, and brings his eyes down to the level of the abdomen, and carefully observes the respiratory waves passing over its surface. After a little experience one is able to detect delicate transverse lines or waves passing upward and downward with respiration. These lines are found to correspond with the curvatures of the stomach.—Ed.]

Palpation of the Stomach.—Inspection is supplemented by palpation, and the results obtained from the former method are corroborated by the latter. Palpation of the stomach is one of the most important, and at the same time one of the most difficult, methods of examination. It should be made a rule to palpate gently; beginners always press too hard, and consequently feel nothing. In the same manner as gentle percussion frequently gives better results than strong percussion, delicate palpation frequently reveals conditions that forcible palpation does not indicate. We need hardly warn beginners from attempting palpation with cold hands, as the abdominal muscles naturally contract as soon as touched.

Palpation should be carried on systematically. In many cases of disease of the stomach palpation will yield nothing, and we should anticipate this. Negative results, however, are diagnostically important. The absence of diffuse or circumscribed pain is always important. If the abdominal walls are too fat or are in a state of tension, palpation is unavailing. In many cases the abdominal muscles become tense as soon as they are touched, and the patient must learn to relax them. The majority of clinicians advise palpation with the finger-tips while holding the hand vertically; it seems to me that it is more correct to palpate in two ways. It will depend on what is desired—whether the physician wishes to palpate the whole stomach in its general outlines, or only certain portions of the organ.

I think the correct method is to palpate in a general way in order to gain general information in regard to the whole organ; this is particularly easy if the organ is dilated and atonic. After the size and the position of the organ have been determined, the different parts of the stomach should be examined; if, for instance, inspection and palpation have shown that in a given case the upper boundary of the stomach is situated two fingers above the umbilicus and the lower boundary three fingers below the umbilicus, no one will consider a tumor found immediately underneath the xiphoid process as a tumor of the stomach. Before palpating the different parts of the stomach it is well, therefore, to determine the general position of the organ by palpation. For this purpose the method with the hand held vertically to the abdominal walls is not to be recommended. I usually begin palpation by placing my hand, flat or slightly bent, upon the abdomen, with the ulnar side downward, and perform a stroking movement from above downward. With a little practice it will be an easy matter, particularly in ectasy, to feel the stomach-walls, as they impart a different and more uniformly elastic sensation than do the walls of the intestine. Sometimes it is possible to palpate the lower boundary of the stomach in this manner. This applies to those cases where simple inspection yields no information whatever in regard to the size, position, or outline of the organ. The hand, as stated, should be slightly bent, so that the palpation is performed with the ulnar side.

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[1] *Deutsch. med. Wochenschr.*, May 1, 1902.

Only in a small proportion of cases of stomach-disease, particularly in ectasies of high degree, can the boundaries of the stomach be determined by palpation, and here only after inflation of the organ with gas or distention by ingesta. It is usually impossible to palpate the boundaries of the empty organ; even if it is possible to determine the boundaries by palpation, it is best to inflate afterward for control.

Whether or not we succeed in determining the dimensions and the position of the organ with one of the methods mentioned, the second method of palpation should be employed, and each portion of the organ should be palpated carefully in order to discover tumors, particularly painful points, etc. In this manipulation the hand may be held vertically to the abdominal walls; the finger-tips should touch the abdomen gently; the pressure should be gradual and carefully increased; the terminal phalanges should gradually be pressed inward, but always very gently. These remarks, of course, only give us some general points of view. It is impossible to describe palpation of the stomach in words: it must be learned at the bedside.

In general this examination is carried on with the patient in the dorsal position and the legs drawn up a little. The physician should be seated on the edge of the bed, and not attempt to perform palpation while standing up. First of all, it is more difficult to examine when standing up, and, besides, it is possible to palpate more gently and to feel better when sitting down, and the manipulations are less painful; any palpation of the abdomen that is performed standing up is inefficient. Sometimes it may be advisable to have the patient lie on his side, or, in particular cases, to occupy the knee-chest position or to stand up. Some forms of tumor that are very motile can be palpated only in the latter positions.

In all the manipulations described one hand should be used,—preferably the right hand,—with the physician seated on the right edge of the bed. In other cases it may be necessary to use bimanual palpation; this is particularly the case in tumors. One hand fixes the tumor in a certain position, either by exercising pressure from behind or in front; in this manner the tumor is forced against the other hand that performs the more delicate palpation, and determines the size, the consistency, the outline, pulsation, painfulness, active or passive motility, etc.

In some cases the examination is rendered difficult by distention of the abdomen or of the intestine with gas. In cases of this kind the bowels should be thoroughly evacuated. . It is never well to rely upon one examination, and the stomach should be examined at different times—before and after eating. Some conditions can be recognized better if the stomach is full; others, if it is empty. Tumors, for instance, of the posterior wall can be felt only when the organ is empty; others again can be felt only when it is full. If patients are always examined at the same hour, many important points may remain undiscovered. Some patients always hold their breath during the examination, and it seems well to request the patient particularly to breathe as usual; deep and slow breathing aids the examination less than normal or slightly accelerated breathing.

All the other abdominal organs should, of course, be palpated in every case of stomach-disease. Dislocation of the kidneys should be particularly looked for. It would lead us too far to formulate the rules of this diagnostic procedure. Occasionally a rectal exploration will be necessary. We need not modify this.

I will refer only briefly to the different points that have to be remembered in this examination, and will recur to them when discussing the different forms of stomach-disease. The first thing to do is to determine whether the region of the stomach is painful or sensitive to pressure. It is well to determine exactly whether the pain is localized in one circumscribed area or is more diffuse; whether it is spontaneous or is felt only on pressure; whether it is increased on pressure, etc. It should be determined whether there is only sensitiveness to pressure that is merely a disagreeable sensation with no real pain, or whether there is pain. In patients that are not intelligent general statements should not be accepted, but the facial expression should be observed; if there is real pain, the slightest touch will cause a grimace. The patients frequently complain of pain in distant parts of the body—for instance, on the corresponding place of the spinal column. The exact location of this pain should be determined, because it may often yield clues for the diagnosis.

Boas[1] has devised an instrument called the algesimeter, that is used to determine the intensity of pain in some given spot. It is constructed analogously to the apparatus used in neuropathology. It consists of a hollow cylinder in which there is a spiral spring; on the outside of the cylinder is a scale that indicates the tension of the spring. Around the cylinder there is another spring that follows the motion of the spiral spring, so that the pressure exercised by compression of the cylinder against the abdomen is registered on the scale. Sometimes two or three smaller cylinders can be attached to the lower portion of the apparatus if it is desired to delineate some larger or smaller painful area. By means of this apparatus Boas discovered, for instance, that round ulcer of the stomach may be painful if a pressure of from $\frac{1}{2}$ to 1 kilo (1 to 2 pounds) is exercised, whereas in chronic gastritis 4 to 5 kilos (9 to 11 pounds) can be borne without pain.

According to Boas, pressure-points on the spinal column are particularly important. There are either pressure-points that are regularly distributed, or more or less extended, irregular, painful areas on both sides of the spinal column. Such conditions are, for instance, found in neuroses of the stomach and intestine. Or there may be strictly circumscribed, unilateral pressure-points, as in gastric ulcer and cholelithiasis. In ulcer, according to Boas, a pressure-point is found in over one-third of the cases to the left of the spinal column, very near the body of the twelfth thoracic vertebra, or a little above or below this bone. In cholelithiasis the painful area is always situated in the region of the twelfth thoracic vertebra or more to the right, some two or three fingers away from the bone. According to my own experience, the pressure-point is frequently absent in ulcer, so that no conclusions can be drawn from its absence.

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[1] *Loc. cit.*, p. 75.

If palpation reveals the presence of a tumor, its position, its size, its shape, its consistency, its painfulness, and its motility should be determined. In many cases the most careful palpation will not be able to determine whether the tumor belongs to the stomach or to some other organ. Inflation of the stomach, which we will describe later on, is of paramount importance in rendering this decision, and is also a valuable means of determining the motility or non-motility of the growth.

I wish to refer briefly to two other points that may lead to error. The one is the possibility of confounding a tumor of the stomach with the pancreas; normally, this organ cannot be felt through the abdominal walls, but if the abdominal muscles are relaxed and thin; if the walls of the stomach are thin and the organ is empty—the pancreas may occasionally be felt in the region of the epigastrium and may be mistaken for a tumor. This can happen still more readily if the stomach is dislocated downward so that the pancreas is exposed over a large area above the lesser curvature (see case described below). In cases of this kind inflation of the stomach will frequently reveal that the tumor does not belong to the stomach, but is situated above the lesser curvature. It has been known for a long time that in thin subjects whose stomach is low down the pancreas can be felt through the abdominal walls and can simulate a tumor.

The second point has been emphasized by Ewald.[1] There is a lymph-gland situated in the middle of the greater curvature in the gastrocolic ligament. In inflammation in or about the stomach this gland may enlarge and become palpable, particularly if the axis of the stomach is rotated in such a manner that the greater curvature is pressed against the abdominal wall. In cases of this kind a circumscribed, small, motile tumor is felt, and if the stomach is dilated, it will be found that this swelling belongs to the stomach. Ewald states that this peculiar structure has frequently complicated the diagnosis and led to the formulation of an incorrect diagnosis.

While it is true that a swelling of this kind may lead us to suspect carcinoma, there are certain points that will help us avoid this error—namely, the peculiar position of the swelling, which does not correspond to the ordinary seat of cancer; the absence of all other symptoms of carcinoma; inflation of the stomach, which will show that this tumor is not situated in any of the favorite locations of carcinoma, but in the middle of the greater curvature, and other signs. Of course, it will be necessary to make repeated examination and to consider all the criteria of the case in order to avoid diagnostic errors.

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[1] *Klinik der Verdauungskrankheiten*, 1893, third edition.

Finally, we wish to call attention to the fact that fecal tumors, particularly if they are situated in the transverse colon, may simulate neoplasms of the stomach. If the case is frequently examined and the bowels are moved by a laxative, such an error should not occur. It should also be remembered that although carcinoma of the pylorus is the most frequent form of gastric cancer, the tumor need not always be in the same place, for carcinoma can be localized in other portions of the stomach, and in tumor the organ is usually more or less dislocated.

Palpation will frequently reveal swelling of the inguinal or supraclavicular glands on the left side. According to my personal experience, these swellings are of no significance in the diagnosis of cancer. These glands are so frequently enlarged in many other diseases that the symptom is of no value in the diagnosis of carcinoma, and, on the other hand, swelling of the supraclavicular glands of the left side is so frequently absent in carcinoma that the symptom is altogether valueless. A positive finding might be of some value. Occasionally small hernias in the region of the epigastrium, that feel like subcutaneous tumors, may be significant, because they may become the starting-point of a number of gastric disturbances.

[The diagnostic importance of Stiller's sign, the presence of the floating tenth rib, has recently attracted considerable attention. A very good study of the question has been made by Walter Zweig,[1] who tabulates 100 cases—50 males and 50 females. Among these he found 49 cases of dyspepsia nervosa. There were 39 cases of enteroptosis, 16 of gastric atony, 27 cases of enteroptosis without atony, 4 cases of atony without enteroptosis. There were various cases associated with gastric conditions of more or less importance. It has been generally accepted that in individuals having a floating tenth rib enteroptosis with accompanying atony and nervous symptoms is relatively common. Beyond this it is hardly safe to go.—Ed.]

Splashing.—Splashing (succussion sound) is heard when air and liquid are shaken together. This sound can never be elicited in an empty stomach. Splashing of the stomach is no pathologic phenomenon *per se*, for it can often be heard in a normal stomach. It is only pathologic if it is heard at a time when the stomach should normally be empty, or if it is heard over an area that is abnormally large and extends beyond the normal boundaries of the stomach. Occasionally both these abnormal features are found together.

Splashing is elicited by tapping the abdominal wall in the region of the stomach. The movement should be performed rapidly, not too forcibly; if too much force is employed, the concussion will be transmitted to the stomach from neighboring organs, and in this manner simulate enlargement of this organ.

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[1] *Boas' Arch.*, vol. vii., No. 3.

Splashing can be elicited in patients with healthy stomachs and relaxed abdominal walls after they have taken large quantities of fluid. In normal subjects, however, these sounds will never be heard outside of the boundaries of the stomach. If ectasy exists, it may be heard over very large areas. The boundaries of the stomach can be determined with considerable accuracy by tapping first from above downward and then laterally, and noting where the splashing stops.

On the other hand, I wish particularly to emphasize that we are not justified in diagnosing dilatation of the stomach or an abnormal size of the organ if we hear splashing below the umbilicus. Kussmaul, several years ago, called attention to the fact that enlargement of the stomach can be diagnosed only under these conditions if dislocation of the organ due to altogether different causes can be excluded.

Splashing heard at a time when the stomach should be empty is very important. It demonstrates that the stomach did not get rid of its contents overnight; in other words, that it is atonic to a great degree. If splashing is heard seven hours after a test-meal or a few hours after a test-breakfast, this indicates motor insufficiency or atony of the stomach, even though the splashing is not heard beyond the umbilical line. If splashing is heard after the ingestion of small quantities of fluid (50 to 100 c.c.—1½ to 3 fl. oz.), this may be considered another symptom of gastric atony. This sign is, however, not absolutely diagnostic of mechanical dilatation with reduction of the motor powers of the stomach-walls.

We see, therefore, that the presence of splashing in abnormal locations determines dilatation of the stomach, and the appearance of splashing at an abnormal time determines atony or motor insufficiency of the stomach. Splashing in itself demonstrates nothing more than that air and fluid are present at the same time. It is pathologic only when it is present in abnormal locations or at the wrong time.

It is easy to differentiate it from gurgling and other sounds that are generated only when the stomach contains air and no water or very little water. They are caused by the tension and the relaxation of the epigastrium following the peculiar manipulations described above (Kussmaul).[1] Splashing can be heard only if the stomach is full or partly so, and others sounds only when it is empty—that is, principally in the morning. It is almost impossible to elicit splashing in any other than the dorsal position or by shaking the whole body when the patient is standing up. The other sounds may be elicited by the pressure of the fingers when the patient is lying down, but the patient is also enabled to produce them himself when he is standing up. It seems impossible, therefore, to confound the two sounds. It should also be an easy matter to recognize splashing that is caused in the colon.

Gastrodiaphany, or Electric Transillumination of the

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[1] "Die peristaltische Unruhe des Magens," *Volkmann's Samml. klin. Vorträge*, 1880, No. 181.

Stomach.—The object of gastrodiaphany is to make the outlines and the form of the stomach visible to the eye by electric transillumination. The method is comparatively complicated, and there are a number of other simpler methods of determining the size and position of the stomach. For these reasons gastrodiaphany is not universally employed. The method essentially consists in introducing an intensely strong electric light into the stomach, so that the boundaries of the organ become visible through the abdominal walls.

Milliot[1] was the first to use electric light for purposes of transillumination. As early as 1867 he performed experiments of this kind on the stomachs of cadavers and animals. Einhorn[2] in 1889 for the first time performed this operation in a human being. Heryng and Reichmann[3] reported a series of exhaustive experiments in this direction that they carried out in 1892. Their apparatus consisted of a soft stomach-tube, at the lower end of which an Edison incandescent lamp was attached. At the upper end of the tube two screws are attached for the wires. In order to increase the intensity of the light the lamp is covered with a glass reflector, and a stream of water is allowed to flow around it in order to prevent burning.

According to these authors, it is necessary that the patient should be accustomed to the stomach-tube, because otherwise gagging and vomiting will interfere with the test.

Heryng and Reichmann recommend filling the stomach with water (500 to 2000 c.c.—1 pint to 2 quarts) and placing the patient in the erect position, because if the stomach is empty, only that portion is illuminated that is situated directly above the lamp. If food or air is present in the stomach, the results are also unsatisfactory. If the light is introduced, a bright luminous area will be seen on the anterior abdominal wall, corresponding in outline and position to the upper and lateral boundaries of the stomach.

According to the experiments of Heryng and Reichmann, the results of this method coincide with those of percussion. One advantage of transillumination is the possibility of determining the upper boundary of the stomach, whereas percussion frequently fails in determining the difference between stomach and intestinal sounds.

Heryng and Reichmann, basing their contention on their experiments, claim that transillumination of the stomach is a method that is serviceable in certain cases for determining the position and the outline of the stomach and the liver.

Einhorn[4] has also published a report on the result of his experiments with transillumination since 1889. His method is somewhat different from the above. He lets the patient drink one or two glasses of water on an empty stomach, introduces the lamp, and examines in a dark room; the patient is allowed to lie down or to stand up. Heryng and Reichmann, on the other hand, recommend the erect position.

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[1] Milliot, Paris, 1867; *Schmidt's Jahrbücher*, No. 136, p. 143.

[2] *New-Yorker med. Wochenschr.*, 1889.

[3] *Therapeut. Monatsh.*, 1892.

[4] *Berlin. klin. Wochenschr.*, 1892, No. 51.

Einhorn uses and recommends transillumination chiefly in the diagnosis of dilatation of the stomach. He claims one advantage for this method that inflation of the stomach with air or filling the organ with water does not possess—namely, that the organ is seen in its natural position, as the small quantity of water (one to two glasses) does not distend the stomach. In inflation and filling with water, on the other hand, the organ is stretched and not seen in its natural state.

Einhorn recommends transillumination particularly for the differential diagnosis of gastric dilatation and gastroptosis; the two are differentiated by the small luminous area seen in gastroptosis as opposed to the large surface that is illuminated in dilatation. In gastroptosis, furthermore, the luminous area is situated low down, and extends about from the umbilicus to the region of the symphysis pubis.

Einhorn mentions one case in which it was impossible to perform transillumination of the stomach, because apparently the walls of the stomach were no longer translucent. He is inclined to interpret this case as signifying thickening of the anterior stomach-wall.

Einhorn considers the chief advantages of transillumination to consist in a rapid diagnosis of dilatation, a positive determination of gastroptosis, and a recognition of tumors or thickening of the anterior wall in cases wherever transillumination does not succeed.

Kuttner and Jacobson,[1] under the direction of Ewald, have published some very thorough and exhaustive experiments on transillumination. These experiments are particularly important, first, because they were carried on in a great many cases; second, because they were in part controlled by postmortem examinations. They used Einhorn's apparatus without the water-cooling arrangement. They claim that circulation of water is not necessary, as Heryng and Reichmann state, and that the stomach is never heated too much. They also state, in contradistinction to the above-named authors, that the introduction of a diaphanoscope is not more difficult than that of the ordinary stomach-tube. The examination can be carried on with the patient in the erect or the recumbent position. A dark room is not necessary, but preferable. Transillumination is best performed when the stomach is empty; if the viscus is filled with food, no picture is obtained, so that it is necessary to remove all coarse particles.

In order to obtain very distinct pictures it is well to fill the stomach with $\frac{1}{2}$ to $1\frac{1}{2}$ quarts of water. In order to do away with the preliminary passage of the stomach-tube for the purpose of introducing this quantity of water, Kuttner and Jacobson have attached a special tube to Einhorn's gastrodiaphane, the opening of which is situated at the lower end of the tube, above the lamp.

At first they used a gastrodiaphane with two tubes, one to allow the water to flow in, the other one to allow it to flow out; later they used an instrument with only one tube. The construction of the instrument will be understood from Fig. 1.

The illumination of the surface of the anterior abdominal wall, according to these authors, corresponds not only to the stomach, but to portions of the intestine that are filled with air and are adjacent to the stomach, as these parts allow a certain portion of the light to shine through. The picture is obscured in part by those portions of the liver that are situated over the stomach, by tumors in the anterior abdominal wall, and by coils of intestine filled with fecal matter that are situated in front of the stomach.

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[1] *Berlin. klin. Wochenschr.*, 1893, Nos. 39, 40.

If the stomach is in a normal position, the luminous area is cut off sharply by the lower margin of the liver, so that we are enabled to determine only the lower and lateral boundaries of the stomach. For this reason gastrodiaphany alone will never enable us to make a diagnosis of gastrectasy.

In gastroptosis all this is different, because a large portion of the organ is situated close to the anterior abdominal wall and is not covered by any other organ, and because the whole organ is dislocated downward. In cases of this kind transillumination yields very characteristic pictures, because it is often possible to determine the upper boundary of the stomach. In addition, the stomach is no longer in contact with the diaphragm, so that the luminous figure is not changed with each respiratory movement. In dilatation the conditions are not so favorable, because the upper part of the stomach remains in its normal position, and is, therefore, not translucent, and at the same time the organ remains in contact with the diaphragm, so that the figure moves with each respiratory effort. If, therefore, the lower boundary of the luminous area moves up and down with respiration, it can be said that the lesser curvature is in its normal place, and if the picture appears below the umbilicus (in case transillumination of the intestine can be excluded), that the greater curvature is situated too low down; the combination of these two findings will lead to the diagnosis of gastrectasy. If the stomach is placed vertically or is distorted, the illuminated area may be lower down; at the same time it will not move with respiration, because the lesser curvature is more or less removed from the diaphragm.

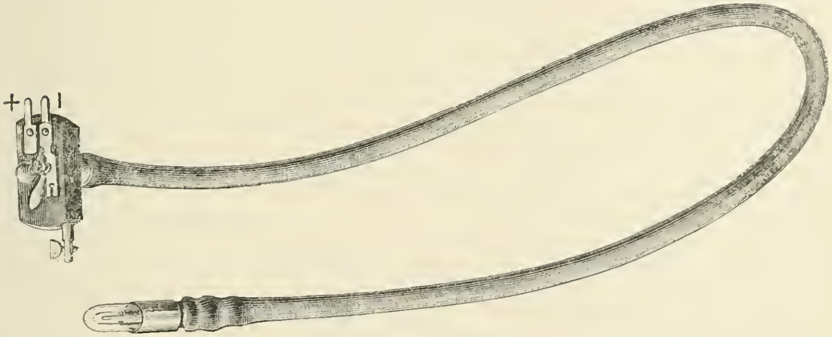

FIG. 1.—Gastrodiaphane.

Tumors of a certain thickness will, of course, not allow any light to pass, so that gastrodiaphany may, under certain conditions, aid in the diagnosis of tumors, sometimes even at a time when neoplasms could not be recognized by any other method of examination. The author has seen this in a number of cases. Here examination with transillumination of the stomach will yield a negative result in case the tumor is thick enough.

According to Kuttner and Jacobson, transillumination is a valuable method for differentiating gastrectasy and gastroptosis. The diagnosis of tumors is also rendered easier.

Meltzing[1] has recently published a most exhaustive report. This author performed a number of experiments on healthy subjects, and arrived at the conclusion that the empty stomach occupies a much larger portion of the epigastric region than is indicated by percussion, inflation, etc.

Meltzing interprets the uncertain results of percussion to signify that this method of examination reveals only the outlines of that part of the stomach that is in direct contact with the anterior abdominal wall. The greater curvature, however, is not in contact with these walls, but is removed from them, and its position can consequently not be determined by percussion. The same, he says, applies to the stomach after it is distended with air or gas or filled with water.

He also contradicts Kuttner and Jacobson, who claim that transillumination permits a differential diagnosis between gastroptosis and ectasy in the sense that respiratory motility of the luminous area is absent in gastroptosis and present in gastrectasis. Meltzing has shown that the respiratory movements of the abdominal organs are not dependent on their direct contact with the diaphragm, but that the most important factor in that respect is the position of the patient. In the dorsal position respiratory motility is pronounced; in the erect position it is very slight. Meltzing does not believe that a differential diagnosis between gastrectasy and gastroptosis can be made from the presence or absence of respiratory motility of the luminous area, as stated by Kuttner and Jacobson.

Meltzing performed his experiments partly when the stomach was empty, partly when it was filled with water. He determined the boundaries of the organ by moving the lamp, and this seems a good suggestion. A comparison of the outlines of the filled organ with those of the empty one will show that those portions of the stomach that are situated in the median line or to the right of it show greatest variations if the organ is filled to different degrees (compare Fig. 2 of Meltzing's, which indicates the average).

Whereas the results obtained by Meltzing seem to overthrow some of our preconceived ideas in regard to the size and the position of the stomach, they cannot refute our general ideas and conclusions. Even though percussion and inflation do not reveal the position of the lowest portion of the stomach, and even though the pictures obtained are too small, this does not alter our deductions, because the pictures obtained in ectasy are also too small. The absolute measurements may be wrong, but the relative proportions are the same, because the error is the same in both cases, and consists in not determining the position of those portions of the stomach that are not in contact with the abdominal walls.

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[1] *Zeitschr. f. klin. Med.*, vol. xxvii., pt. iii., iv.

Meltzing formulates one other conclusion that is interesting—namely, that gastrectasy can never be diagnosed by transillumination alone, because a perfectly normal stomach may show similar boundaries.

My opinion is that although gastrodiaphany has given us so many interesting results, there is little prospect of its being universally employed. From a scientific point of view the procedure is unquestionably valuable; practically we can get along very well without it. As long as this method remains as complicated as it is and the appliances needed are so numerous and bulky; as long as the results obtained from this method are so uncertain and unpractical—we can hardly expect that the method will be generally employed. In those cases where the diagnosis must be made at the bedside we can get along without this method. Again, it must be remembered that gastrodiaphany frequently furnishes a very incomplete picture of the outlines of the stomach, and at times gives absolutely negative results. Features that will prevent the universal application of this method in practice are the expense of the apparatus, the necessity of having an assistant, and still other factors. It will usually be possible to make a differential diagnosis between a gastrectasy and gastroptosis by some simpler method. Those cases in which the diagnosis of a tumor can be made by transillumination before its presence is revealed by other methods are very rare, and in cases of this kind, moreover, the picture obtained by transillumination is capable of various interpretations.

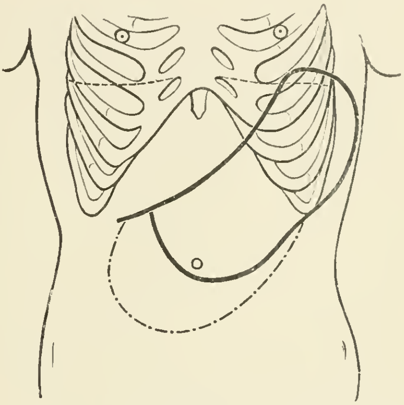

Fig. 2.— ━━ Transillumination picture of the normal stomach when empty. ---- Transillumination picture of the normal stomach when full.

[Bade[1] found that the inflated stomach was much more permeable to the Roentgen rays, and that by inflating with air or CO_2, a skiagraph of the stomach could be obtained.

The *x*-ray method has been further utilized in studying the movements of the stomach by observing the shadows produced by bismuth, mixed with the ingesta, or made into small pellets with masses of soft bread. Some very interesting studies in this direction were made by W. B. Cannon.[2] The same observer has recently published some equally interesting results from his studies of intestinal peristalsis.—Ed.]

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[1] *Deutsch. med. Wochenschr.*, No. 38.

[2] *Jour. Boston Soc. Med. Sciences*, Feb. 15, 1898.

Gastroscopy.—In contradistinction to gastrodiaphany, gastroscopy aims to illuminate the interior of the stomach and to make it visible to the eye of the observer. Mikulicz[1] has done some very creditable work in introducing gastroscopy and in making it practical. Mikulicz's gastroscope consists of a thick, stiff metal tube 22 inches long and $\frac{9}{16}$ inch thick, composed of three tubes—one for the wires, another for water, and the third for inflation with air. At the gastric end of the tube an electric lamp is attached. The picture of the portion of the mucosa that is illuminated is reflected through several prisms. The apparatus is connected with a bellows, so that the stomach can be distended or inflated. The water-tube is intended to allow the circulation of water in order to prevent heating of the gastric mucosa.

Mikulicz has demonstrated that this apparatus yields positive results. In a number of cases he succeeded in diagnosing carcinomata and ulcers by this method. It would lead us too far to describe the details of the gastroscopic pictures observed. However interesting the results obtained by Mikulicz with this instrument, the method will not be universally employed, owing to the great technical difficulties of executing it. The manipulations are difficult, disagreeable to the patient, and the apparatus is very expensive. We can hardly expect that this method will be more generally employed until the apparatus has been simplified.

Percussion of the Stomach.—Percussion of the stomach can determine the size, the boundaries, the tension, and the character of the contents of the stomach. As a rule, however, it is impossible to determine all these factors. The determination of the size and the extent of the stomach is a very difficult matter; this is due to the fact that much will depend on the amount of material contained in the stomach, and that the stomach is never completely in contact with the anterior abdominal wall if it is distended. As a rule, in this condition it is covered by other organs. Finally, percussion is rendered difficult because the stomach is in contact with other organs that contain air. The stomach can be differentiated from other portions of the intestines only by percussion where two adjacent parts have different contents, so that they give a different sound. Quite frequently it is altogether impossible to determine the boundaries of the stomach by percussion, even if the plessimeter is used.

The percussion-sound over the stomach will vary according to the contents of the organ—that is, whether it is empty, filled with air, or contains more or less food. Much will depend on the time of examination—that is, whether the stomach is empty or full or contains air; or on the position of the patient—that is, whether he is standing up or lying down. In order to obtain any results whatever from percussion, the stomach should contain some air. If the stomach is empty, a tympanitic or a loud sound will be elicited in the epigastric region below the liver, and heart-dulness, and underneath the lower margin of the left lung. This sound is not produced by the stomach, but by the colon, which is filled with air, for in health the stomach, when it is empty, is contracted and is hidden away in the left concavity of the diaphragm, so that it is nowhere in contact with the anterior thoracic wall, and is altogether out of reach of percussion (Dehio).[1]

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[1] *Wien. med. Wochenschr.*, vol. xxxiii., p. 748.

Owing to this contraction of the stomach a space is created in the epigastric region between the liver, the spleen, and the anterior abdominal wall that must be filled by some other abdominal organ. As a rule, the transverse colon and the left colic flexure move in here. Dehio determined, by examinations of cadaver and of living subjects, that the stomach, when it is empty, never contains a sufficient quantity of gas to cause its distention to such a degree that it touches the anterior abdominal walls. His findings are corroborated by the anatomic investigations of Luschka and Braune. It is not immaterial, therefore, at what time percussion of the stomach is performed. The whole extent of the organ can never be determined by percussion, but only the boundaries of that portion that is in direct contact with the anterior abdominal or thoracic walls and is not covered by other organs.

That portion of the stomach, the position of which is particularly interesting,—namely, the lower boundary, the greater curvature,—is not in contact with the anterior abdominal wall, and it is curved in such a manner that it can never be determined by percussion. In percussing the stomach we usually attempt to determine four boundaries—the upper, the lower, the right, and the left. From a practical point of view the determination of the lower boundary is the most important, because we aim chiefly to determine the size or the extent of the organ by percussion, and the enlarged organ principally extends downward. At the same time we are not justified in diagnosing an abnormal distention of the organ when we find the greater curvature, or, better, the lower boundary of the stomach, low down. The determination of one boundary is of no value whatever in pathologic cases. Simple dislocation of the stomach downward, gastroptosis, and a vertical position of the stomach may cause the lower boundary of the stomach to be found low down, and at the same time the organ will not be enlarged.

According to the exact investigations of Pacanowsky,[2] who examined 81 healthy subjects and patients who were afflicted with stomach-disease, the upper boundary of the stomach is usually situated as follows: in the left parasternal line, at the lower margin of the fifth rib or in the fifth intercostal space, rarely one intercostal space higher or lower; in the left mammary line, in the fifth intercostal space to the sixth rib; in the anterior left axillary line, at the lower margin of the seventh or on the eighth rib, rarely below the sixth rib. He examined his cases in the dorsal position while the stomach was empty or two or three hours after the principal meal.

The right boundary of the stomach can be determined only in its upper portion. It is situated about 2 inches from the median line. The left boundary, according to Pacanowsky, cannot be determined throughout its whole extent; the point that can be determined with the greatest certainty is the point of junction between the upper and the left boundary of the organ, as this point is situated at the uttermost margin of tympanitic stomach-sound to the left, in the anterior axillary line, somewhat below the seventh or on the eighth rib.

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[1] *Verhandl. d. VII. Cong. f. innere Med.*, 1888.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xl.

The lower boundary of the stomach, as we have said, is that most important. It is not always possible, by percussion in normal subjects, to determine this boundary. In order best to do this, it is well to percuss gently in the dorsal position in order to differentiate the colon from the stomach. The result obtained is then controlled by causing the patient to stand up, and finding the beginning of any dulness that may be present. This, of course, is possible only when the stomach is not empty. According to the examinations of Pacanowsky, this lower boundary is found in the left parasternal line—in men some 3 to 5 cm. (1.18 to 2 in.); in women some 4 to 7 cm. (1.57 to 2.75 in.), above the umbilicus. The greatest height of the stomach found in man is from 11 to 14 cm. (4.3 to 5.5 in.); in women, usually 10 cm. (4 in.). The percussion-sound over the colon rarely equals that over the stomach, and a lighter sound is usually heard over the colon when the patient is lying on his back.

Pacanowsky recommends the introduction of small quantities of carbonic acid into the stomach in order to control the sound, and to differentiate the tympanitic sound of the stomach from that of the intestine. The small quantity of carbonic acid gas that Pacanowsky introduces does not change the boundaries of the stomach; even the lower boundary of the stomach remains at the same height. The lower boundary of the stomach is usually found 3 to 6 cm. (1.18 to 2.36 in.) above the umbilicus, rarely lower down, so that those cases in which the lower boundary of the stomach is below the navel must be considered pathologic.

Martius[1] and Meltzing,[2] from their investigations with diaphanoscopy, state that the lower boundary of the stomach is normally lower down than is usually assumed, and that the outline presented by transillumination is different from that delineated by percussion. They claim that the stomach, when empty, reaches as far as the umbilicus, and when it is filled, still further down. According to Martius, the lowest point of the stomach boundary can be found on a line drawn between the two highest points of the crests of the ilia. In the erect position this boundary is moved 3 to 4 cm. (1.18 to 1.57 in.) downward.

We do not know as yet to what extent these results will modify our views on the size and the position of the stomach. We have already called attention to the fact that percussion yields figures that are too small, because percussion can determine the extent only of those portions of the stomach that are in direct contact with the abdominal wall, and never the extent of the whole organ. For this reason all measurements obtained by this method are too restricted. At the same time the values obtained by percussion are useful, inasmuch as they give correct relative measurements, even though the absolute measurements are not correct.

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[1] *Verhandl. d. 66. Naturforscherversamml.*; see also an abstract in *Münch. med. Wochenschr.*, 1894, No. 41.

[2] *Zeitschr. f. klin. Med.*, vol. xxvii., Nos. 3, 4.

The determination of the size of the stomach *per se* is devoid of much value; a determination of its motor powers is much more important from a practical point of view.

It will be seen that the determination of the size of the stomach by percussion is frequently difficult; for this reason other methods have been sought to determine the outline and the shape of the organ. It was particularly important to determine the exact boundaries and shape of the stomach in cases where the organ was dilated, and wherever it was necessary to differentiate dilatation from an abnormal position of the undilated viscus. We will discuss the different methods that have been evolved for this purpose in the following chapters.

Artificial Inflation of the Stomach with Carbonic Acid Gas.—Frerichs, in Germany, was the first to describe methods for inflating the stomach with carbonic acid gas for the purpose of determining its form and extent more exactly by percussion. Samuel Fenwick[1] was the first to employ this method in England. The latter, in 1868, advised examination of the stomach before eating, and again when the organ was dilated, in order to avoid confusion between the stomach and the colon on percussion. He produced the dilatation by giving the patient some soda-water or an effervescent powder. Wagner,[2] in 1869, reports some similar experiments on inflation of the stomach with carbonic acid from Mannkoppf's clinic. The dose he gave was small as compared to the doses we give to-day, as he administered only a pinch of tartaric acid and of bicarbonate of soda. He also recommended another similar procedure—namely, the drinking of a glass of artificial soda-water.

Wagner, as well as Fenwick, desired merely to determine the boundaries of the stomach more accurately by percussion, so that these small doses were sufficient. According to Wagner, this artificial distention of the stomach with carbonic acid gas causes the creation of a column of gas of a greater diameter than is ever found in any other portion of the intestinal canal, and the tympanitic sound over the whole extent of the organ becomes more tympanitic than over any other portion of the intestinal tract.

Even of late years Boas has recommended small doses of from 1 to 2 grams of tartaric acid and of sodium bicarbonate. I am in the habit, as I will show presently, of employing much larger doses. Von Ziemssen[3] and Ebstein[4] do the same. I usually administer a teaspoonful of bicarbonate of soda and a little less tartaric acid; each salt is dissolved in about half a glass of water. The tartaric acid solution is taken first, and then the soda solution. We use large doses because we do not simply wish to make the outlines of the stomach more distinct for percussion, but because we wish to make the outlines of the organ both visible and palpable. Before inflation it is necessary, of course, to examine the stomach for tumors. In case they are present the clinician should determine exactly how they are situated and what their consistency is.

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[1] *The Morbid States of the Stomach and Duodenum*, London, 1868.

[2] *Inaug.-Diss.*, Marburg, 1869.

[3] *Klin. Vorträge*, 1888, No. 12.

[4] *Volkmann's Samml. klin. Vorträge*, 1878, No. 155.

In many cases the stomach will be seen to protrude immediately after the administration of the effervescent powder. The large curvature in part becomes clearly visible, whereas the lesser one is not so distinct or remains altogether hidden. Even though the dilatation of the organ be not great enough to make its outlines visible, palpation may frequently reveal the upper and lateral boundaries. Naturally, percussion of the organ is rendered more exact by this procedure, as the area of tympanitic sounds corresponds to the boundaries of the stomach.

In case tumors are found before inflation is performed it is important to determine their position after inflation. In many cases this double examination will show whether the tumors are connected with the stomach or not, or whether they are motile or fixed. When inflation is performed, the stomach is not only distended, but also rotated around its longitudinal axis. In this manner it may happen that tumors that are situated to the right and on a level with the umbilicus when the stomach is empty, move upward and to the right, toward the anterior costal arch, when the organ is inflated. This will determine their connection with the stomach, particularly as it is often possible to see and to feel a direct connection between the tumor and the stomach. It may even be possible to demonstrate that it is near the lesser curvature or near the pylorus; in other cases it will be an easy matter to discover that the tumor does not belong to the stomach. If a tumor disappears or becomes less apparent after distention of the stomach with carbonic acid gas, we can usually assume that the growth appertains to the posterior wall of the organ. If the growth is freely movable, the presence of solid adhesions is excluded; if the growth is altogether immovable, we can diagnose abnormal fixation.

We see, therefore, that inflation of the stomach yields more information than the original discoverers of this method suspected. All that these clinicians desired was a method to determine the boundaries of the stomach with more facility and to differentiate the organ from neighboring parts that were filled with air. In order to extend the value of this method in the manner described in the preceding paragraph, large doses should be given.

In very obese individuals the method fails; this can hardly be considered a serious objection to its employment. There are, however, other more valid objections. Runeberg,[1] for instance, calls attention to the fact that it is impossible to control the degree of tension, so that occasionally the carbonic acid gas may irritate the stomach and produce disagreeable concomitant symptoms. Pacanowsky[2] concedes that carbonic acid is the best gas for distending the stomach; at the same time he calls attention to the fact that very large doses might distend the stomach too much. He claims that the introduction of carbonic acid gas into an empty stomach yields nothing, whereas the same amount given after eating distends the organ considerably.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxiv.

[2] *Ibid.*, vol. xl.

Whoever has performed inflation frequently will agree with von Ziemssen,[1] who says that the stomach-walls can be distended only to a slight and limited degree, with the exception possibly of cases of pathologic thinning of the stomach-wall and degenerative atrophy of the muscularis. He also claims that as soon as the pressure grows too great, a part of the gas will escape upward through the cardia. To judge from my own experience, dilatation never becomes too great, and consequently the size of the stomach is never exaggerated. I have never so far seen any disagreeable consequences among thousands of inflations that I have performed. I wish to emphasize again that the value of the method does not consist alone in determining the lower boundary of the stomach, but also in delineating the form and position of the stomach, and establishing the presence or absence of tumors, etc., that may be connected with the organ. No other method yields such positive information in regard to the differential diagnosis between ectasy and vertical position or gastroptosis as inflation. The fact that inflation of a normal stomach does not cause protrusion of the whole organ, but principally of the lower half, can hardly be considered an objection to this method.

Inflation with carbonic acid gas is not dangerous. If desired, the stomach-tube may be held in readiness for emergencies; I always do this. If inflation causes too much distress, the tube can be introduced and untoward symptoms immediately relieved. Personally I have never seen any disagreeable consequences or any symptoms whatever, although I have performed inflation almost daily for many years.

The question arises whether we are justified in employing inflation with carbonic acid gas in every form of stomach-disease. There are undoubtedly cases in which the method is contraindicated—for instance, where hemorrhage has recently occurred, where there are signs or a suspicion of ulcer, or symptoms of peritonitic irritation. The indications for the employment of this method are self-evident from what has been said; it is indicated wherever other methods do not give us sufficient information in regard to the position, the size, and the outline of the stomach, particularly in those cases where there is a suspicion of ectasy, gastroptosis, dislocation, or abnormal position or fixation of the organ, and wherever none of the above-named contraindications exist. [In the inflation of the stomach by carbonic acid gas there is an element of danger in case of ulcer, carcinoma, or other disease that seriously weakens the gastric walls. It is true that accidents rarely occur, and yet caution should be practised when the walls of the stomach are known to be diseased. Behrand and Hughes[2] report three fatal accidents resulting from the use of this method.—Ed.]

Whereas, in general, the administration of an effervescent powder causes dilatation of the stomach alone, we occasionally see that other parts of the abdomen are distended at the same time. Here the tension of the stomach-walls is slight, but at the same time there is tympanitic sound over the intestines that may vary in intensity in different parts of the abdomen, depending on the degree of tension in the different parts of the intestine.

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[1] *Klin. Vorträge*, 1888, No. 12.

[2] *Med. News*, June 14, 1902.

These peculiar symptoms, according to Ebstein,[1] who first called attention to this state, are due to incontinency of the pylorus. We do not know at present what they are due to in each individual case, and whether they are caused by some anatomic lesion or are of nervous origin. At all events, we know that in the majority of cases the gas immediately escapes through the pylorus. According to Ebstein, this symptom is seen in a number of diseases, most frequently, however, in ulcerative disturbances or infiltration of the pylorus. Ewald claims to have seen this insufficiency very rarely, and says that when it does occur, it is caused as follows: Dilatation of the stomach with carbonic acid gas causes spastic closure of the cardia, and the patient is stimulated to expel the gas. The pylorus is more liable to yield than the cardia, consequently the gas enters the small intestine. Ewald is of the opinion that in those cases where the pylorus does not yield at once, but only after the development of carbonic acid gas is well under way, the first-named condition exists. It appears, therefore, that Ebstein's insufficiency of the pylorus is not very important. As far as my own investigations go, this explanation is hardly valid. In the case that I observed, the pylorus did not yield after the lapse of some time, but the gas escaped at once, so that there was no distention of the stomach. Normally, the gas escapes upward; less readily downward. Those patients in whom distention can be performed without difficulty prefer to get rid of the gas by belching, instead of waiting until the pylorus opens the way downward. I am inclined, therefore, to consider the escape of gas through the pylorus as pathologic. I can corroborate Ebstein's observation, although it may be difficult in each individual case to determine the exact cause of this pyloric insufficiency without the assistance of other methods of examination.

Inflation of the Stomach with Air.—Instead of inflating the stomach with carbonic acid gas a method of inflating the organ with air has of late been recommended. An ordinary soft stomach-tube is introduced and connected with a double bulb, like the one on Richardson's spray. Air is then slowly pumped into the stomach.

Inflation with air is performed for the same purpose as inflation with carbonic acid gas, and the same indications and contraindications exist for this procedure. Runeberg[2] was the first to recommend this method in place of the carbonic acid method. This author believed that it possessed certain advantages over the older method; he claimed that it did away with a number of objectionable features—for instance, our inability to determine the exact degree of tension, the occurrence of disagreeable concomitant symptoms caused by the irritating effect of gas on the mucosa, the retention of carbonic acid gas and of some of the salts in the stomach.

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[1] "Ueber die Nichtschlussfähigkeit der Pylorus," *Samml. klin. Vorträge*, 1878, No. 155.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xxxiv.

Runeberg recommends inflation of the stomach with air in the same manner as inflation of the colon is performed. He claims that his method allows distention of the stomach or of the colon to any degree, and sees an additional advantage in his ability to release the air or to pump more air into the organs at will. The disadvantage of the method is the introduction of a tube.

The method of Bouveret[1] is still more simple; he recommends blowing the air from the mouth directly into the stomach-tube; if any one is squeamish about placing his lips to the free end of the tube, he can insert a small glass tube. All that is necessary to prevent exit of air from the stomach is to pinch the tube between the fingers. As soon as the examination is completed, the fingers are relaxed and the air is allowed to escape.

We must concede that these methods are more exact than the development of carbonic acid gas. At the same time I cannot agree with those who claim that the development of carbonic acid gas within the stomach causes disagreeable symptoms by the direct irritant effect of the gas. As I have said, I have never observed any such symptoms among many thousands of cases.

I have failed to discover any objectionable features in my method. Here and there the result is unsatisfactory, because the dose of powder is too small. This error, however, can easily be corrected when the experiment is repeated. The advantage of this method is that it can be carried out without any instruments and without assistance. For the practitioner, these advantages cannot be overestimated, for the simpler the diagnostic adjuvant, the more readily will it be employed in practice. Inflation of air requires an assistant; if it is desired to make careful examinations, the region of the stomach should be inspected continuously before and during and after the inflation of air. This, of course, can never be done if the observer is introducing the tube himself and blowing air into it. Possibly he may succeed in subjects who are accustomed to the sound, but certainly not in those patients who react to the introduction of the sound with violent gagging.

If plenty of time is at the disposal of the physician, the patient may first be accustomed to the sound, and inflation of air may be practised at some later time; if it is desired to gain information at once, inflation with carbonic acid gas should be performed, and in the majority of cases this procedure will yield sufficient information in regard to the size, the position, and the outline of the stomach, as well as its relation to any tumors that may be present.

I have no personal experience in regard to the method of inflating with air by blowing; one of the chief disadvantages, it appears to me, is the fact that the region of the stomach cannot be observed while blowing air into a stomach-tube. Personally, this defect does not seem sufficient to condemn the method; but, in addition, I dislike to contemplate the possibility of the patient's suddenly gagging, so that I prefer not to employ this procedure, particularly as better methods that do not include these defects are at my disposal. The patient, too, should be considered, and many persons will certainly object to having the physician's breath blown into their stomach.

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[1] *Traité des mal. de l'estomac*, Paris, 1893.

Taking everything into consideration, I still maintain that the method of inflation by carbonic acid gas is practically the simplest. In many cases, particularly in clinics and in hospitals, the method of inflation by air may be employed to advantage.

I have been in the habit of employing both methods for several years. In cases where the patients tolerate the introduction of the sound, and where assistance can be had, the inflation of air is probably preferable. The physician in general practice, however, will find the administration of an effervescent powder more practical. In many cases it may be necessary to distend the colon, particularly if the attempt is made to differentiate the stomach from the colon, or to determine the location of certain tumors in this or the other portion of the intestinal tract.

I have already mentioned that Runeberg recommended insufflation of the colon from the rectum, and carries it out in the same way as inflation of the stomach. Von Ziemssen[1] attempted inflation of the colon before Runeberg; he used a somewhat different method, and introduced bicarbonate of soda and tartaric acid into the colon. According to him, 20 grams of sodium carbonate and 18 of tartaric acid are necessary for this. It is an easy matter to dilate the whole colon by either of the above methods.

Boas[2] has described another method of determining the size and the position of the colon. The intestine is thoroughly evacuated, and water introduced into the colon through a Hegar funnel. After 500 to 600 c.c. (17 to 20 fluidounces) of water have been introduced, all parts of the colon are filled, and splashing can be heard in all the locations that correspond to the normal position of the transverse colon. In the region of the ascending and descending colon a slight amount of splashing can also be elicited. In certain pathologic conditions the introduction of very small quantities of fluid will frequently enable us to obtain splashing; this points to atony of the colon. Or, again, splashing of the transverse colon and of other portions of the intestine is heard in abnormal positions so that dislocation of the colon can be diagnosed. This method is certainly valuable for the diagnosis of atony and dislocation of the colon. For the diagnosis of stomach-lesions Runeberg's method of insufflation of air with bellows is better. In complicated or difficult cases inflation of the stomach with carbonic acid gas may be combined with the method of filling the colon with water (Minkowski[3]). This combined method is particularly valuable in the diagnosis of abdominal tumors in deciding the question whether they belong to the stomach or to some other neighboring organ.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii.

[2] *Diagnostik und Therapie der Magenkrankheiten*, third edition, 1894, p. 101.

[3] *Berlin. klin. Wochenschr.*, 1888, No. 31.

A rule has been discovered that applies to this combined method—namely, that if the stomach and intestine are filled, abdominal tumors move toward the place in the abdomen normally occupied by the organ to which the tumor belongs (Minkowski). The combined method is very valuable in the diagnosis of circumscribed tumors of the pyloric region. When the stomach is distended, tumors of the pylorus usually move downward and to the right. Tumors of the transverse colon and the mesentery may move in the same direction.

If the colon is filled with water, tumors of the stomach in these cases move upward and can frequently be distinguished from the filled transverse colon. Tumors of the lesser curvature move upward and backward and can no longer be felt (Minkowski).

In cases in which the tumor involves several organs the different parts of the growth may frequently be forced away from each other, and the discovery be made that they belong to several organs.

Other Methods for Determining the Size, Position, and Capacity of the Stomach.—Numerous other methods have been described for determining the size, position, and capacity of the stomach. None of them, however, is preferable to inflation of the stomach with carbonic acid gas or with air. The majority of them are not so good. Nearly all of them call for the introduction of the stomach-tube. It would lead us too far to enumerate all the methods and modifications that have been described. We will limit ourselves to a brief description of the most important ones.

(*a*) **The Method of Penzoldt.**—Penzoldt[1] introduces a definite quantity of fluid—about one quart—into the stomach through the tube, and by percussion determines the outlines of the dulness in the area of the epigastrium. Prior to filling the stomach with water this zone was tympanitic. He proves conclusively that this area of dulness corresponded to the stomach by removing the liquid and causing the dulness to disappear.

This method has certain disadvantages. In the first place, it necessitates the employment of the sound; in the second place, it does not determine the volume, form, and size of the stomach, but only its lower boundary. It is impossible by this method to exclude ectasy or dislocation of the stomach.

(*b*) **Method of Leube.**[2]—This author introduces a stiff sound into the stomach and determines the position of its lower end through the abdominal walls. In this manner the location of the lower boundary of the stomach is established. In the course of this experiment he usually found that the lower stomach-wall extended downward as far as the umbilicus. At the same time ectasy should not be diagnosed if the point of the sound is below this level. Gastroptosis or a vertical position of the stomach, without an increase in the size of the organ, may produce this result. This method, of course, yields no information in regard to the size, position, or outline of the stomach; in many cases, moreover, it is impossible to palpate the end of the sound through the abdominal walls. Leube himself has abandoned this method.

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[1] *Die Magenerweiterung*, Erlangen, 1875.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xv.

(*c*) **The Method of Purjesz.**—Purjesz's [1] method differs slightly from that of Leube. In order to determine the lower boundary of the stomach he connects the sound with a manometer. As soon as the sound enters the stomach from the esophagus the manometer, which up to this time shows negative pressure, suddenly shows positive pressure; this occurs at the moment the sound engages the cardia. The sound is now pushed on until an obstacle is encountered. The straight diameter of the stomach from the cardia to the large curvature is expressed by the length of the sound introduced from the time of the first beginning of positive pressure to the time when the obstacle is encountered. The same objections can be formulated against this method as against that of Leube.

(*d*) **The Method of Schreiber.**[2]—Schreiber introduces a sound into the stomach at the end of which a rubber-bag (Victoria bag) is attached. He inflates this little balloon, and in this way attempts to obtain information in regard to the size and the position of the organ. This method has not been universally adopted, and the same results may be obtained in a much more simple manner by insufflation with air.

(*e*) **The Method of Rosenbach.**[3]—Rosenbach claims that in subjects with fat and tense abdominal walls all the foregoing methods are uncertain, and that in very thin subjects the reflex tension of the abdominal muscles will interfere in the same way as fat interferes in obese persons. He believes that all methods that are directed toward determining the outlines of the stomach by pressure on its walls, by fluid or gas, can at best yield relative results. Rosenbach, therefore, attempted to discover a direct measurement for the expulsive powers of the stomach. He assumed that the determination of the level of fluid contained in the stomach could yield certain information in regard to the resistance offered to fluid distending the fundus. His criterion was the rise and the fall of the level of this fluid. If a rubber bag is connected on the one side with the funnel end of the stomach-tube and on the other with an open rubber tube, compression of this ball will enable us to pump large or small quantities of air into the stomach, provided the free tube is closed. If the ear is placed over the region of the stomach, a gurgling, moist metallic sound will be heard, followed by a distinct splashing as soon as the opening of the stomach-tube dips into the fluid contained in the stomach. This sound is caused by the air passing through this fluid. As soon as the sound is withdrawn from the fluid, splashing, of course, stops.

This experiment is carried on as follows: The stomach must be empty; 50 to 100 c.c. ($1\frac{1}{2}$ to 3 fluidounces) of fluid are poured in, and the rubber ball compressed. A distinct gurgling sound will be heard.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxiv.

[2] *Ibid.*, vol. xix.

[3] *Volkmann's Samml. klin. Vorträge*, 1878, No. 153.

In healthy subjects it is necessary to withdraw several centimeters of the sound before the gurgling stops. In this way a fairly accurate measure of the height of the fluid in the stomach can be obtained by measuring the length of that piece of the sound that protrudes beyond the teeth at the time when splashing begins.

Rosenbach has shown that in dilatation of the stomach much larger quantities of fluid are necessary in order to cause a slight rise in the level of the fluid than in normal subjects.

This method does not furnish information in regard to the size of the stomach, but chiefly in regard to its resistance to distention. We are not justified, however, in identifying a decrease in this resistance with a decrease in the motor powers of the stomach. In order to determine the latter, the method of Leube, which we will discuss below, is certainly the best.

(*f*) **The Method of Neubauer.**[1]—This is a very simple one. All that is needed is the apparatus commonly used in washing out the stomach—namely, the sound, a glass funnel with a long tube. The sound is introduced, the funnel filled with water, and the surface of the fluid within the stomach determined by raising and lowering the funnel. If the funnel is in a certain position, the level of water will be in a state of equilibrium, and, according to the law of communicating tubes, will correspond to the level of the fluid present in the stomach.

Rosenbach has experimented in the same direction and independently of Neubauer. The same objections can be made against this method as against the one above.

(*g*) **The Method of Jaworski.**[2]—This method aims at determining the capacity of the stomach, the vital contractility and distention of the walls of the stomach. A special apparatus has been devised for this purpose, with a stomach volumeter (see Fig. 3).

The apparatus is constructed as follows: *A*, a graduated bottle containing at least 6 quarts; it is placed in an elevated position. A tube, *m*, is attached to the bottom of this flask. To this tube there are attached a rubber cork, a glass and a rubber tube, *m*, *p*, *c*, which lead to the bottom of bottle *H*, the capacity of which is as great as that of bottle *A*. The bottle *H* is closed by a rubber cork with three holes; through one of these holes passes the connection with bottle *A*; through the second one a thicker glass tube that is open at both ends (the manometer tube); this is divided into centimeters. Through the third opening passes a bent glass tube, *d*, *n*, which connects with bottle *H* and the stomach-sound *S* through a rubber tube *n*, *g*. Communication between bottles *H* and *A*, as well as between *H* and *S*, can be interrupted by the clamps *b* and *f*.

The experiment is carried out as follows: Measured quantities of water are poured into *A* and *H*—into *A*, about 6 quarts; into *H*, ½ to 1 quart. The sound is then introduced into the empty stomach, and the contents of the stomach are aspirated through a pump, so that all gas and fluid are removed. The clamp *f* is then closed, and the sound connected with the stomach volumeter. The clamps *f* and *b* are then opened, and water allowed to flow from *A* until the patient begins to complain of a feeling of tension on the left side. The clamps *b* and *f* are then closed, and the height of the water column in the tube *e*, *d*, is read from the level of the water in bottle *H* to the top of the column. The capacity of the stomach is indicated by the amount of water that flows from flask *A*, or the increase of water in flask *H*.

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[1] *Prager med. Wochenschr.*, 1878, No. 4.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xxxv., p. 83.

Jaworski lays particular stress on emptying the stomach of all gas and fluid, so that subjects who are not accustomed to the stomach-tube or are inclined to cough are not suitable ones for this method. More than in any other method the objection can be made that this one is too complicated to warrant its introduction into practice.

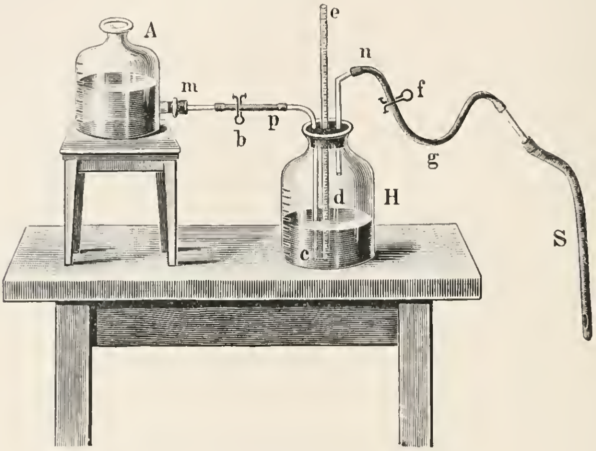

FIG. 3.—Stomach volumeter of Jaworski.

(*h*) **The Method of Kelling.**—Kelling[1] does not consider the determination of the size of the stomach by insufflation of air an accurate one, because the estimation is made from the amount of protrusion of the abdominal walls and because the resistance that the stomach-walls offer is neglected. Kelling, therefore, invented a different method, and used the following apparatus (see Fig. 4). Two T-tubes (α and β) are inserted into an ordinary stomach-tube. The free branch of the first one is connected with a double bellows, *d*, by a rubber tube. The free branch of the second T-tube (β) is connected with a water manometer, *e*. The free end of the stomach-tube is connected with a U-tube *c*, the point of which is drawn out. A graduated measuring cylinder containing 2 or 3 quarts is inverted in a bucket or a vessel of water, *f*, and the free end of the U-tube introduced into this cylinder to a point where it extends above the level of the outer water surface. Cocks are attached near the bellows and near the end of the stomach-tube.

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[1] *Deutsch. med. Wochenschr.*, 1892, Nos. 51, 52.

A test-meal is administered, and the stomach washed out until the water returns perfectly clear. The cock in front of the bellows is then opened, and the one in front of the measuring cylinder closed. Air is then pumped into the stomach until a feeling of pain and distention is complained of; when this point is reached, the cock in front of the bellows is closed, and the one in front of the cylinder opened so that the air passes from the stomach into the measuring cylinder. A certain amount of water is displaced by the air, and the amount of air that was present in the stomach can be calculated from this.

Several additional features must be considered—for instance, the barometric pressure, the water tension, the temperature of the water, of the air that came from the stomach, etc. If it is desired to avoid all this calculation, the volume of air in the cylinder can simply be commuted into the volume of air in the stomach. In order to do this Kelling has determined that some 7 to 8 per cent. must be added to it. It is doubtful whether this complicated procedure will ever be introduced into practice.

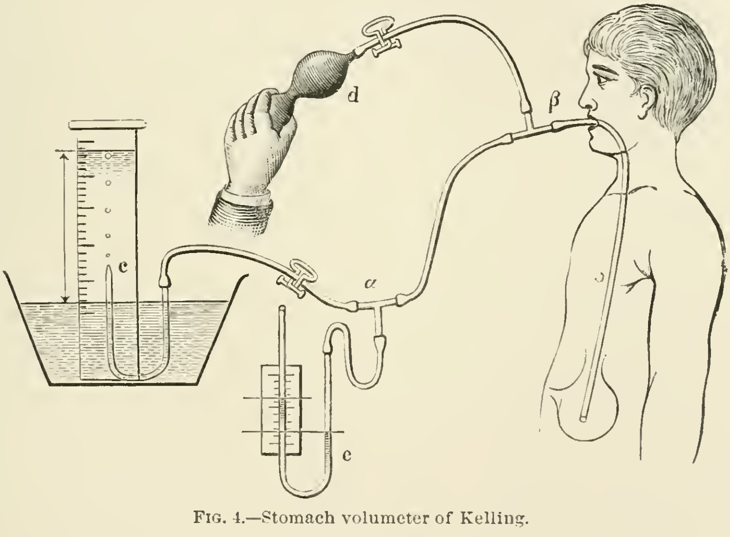

FIG. 4.—Stomach volumeter of Kelling.

(*i*) **The Method of Unverricht and Ost.**—This method, like the ones of Jaworski and Kelling, aims at determining the capacity of the stomach. It is really a modification of Jaworski's method. Ost[1] has described it at great length. The apparatus consists, first, of a stomach-pump that is arranged so that control of the piston allows the aspiration of 150 c.c. (5 oz.) of air into the pump; second, of a mercury manometer to determine the pressure within the stomach; third, a measuring apparatus to gather the air coming from the stomach; fourth, a stomach-sound.

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[1] "Beiträge zur Bestimmung der Capacität des Magens," *Gesammelte Abhandl. aus der med. Klinik in Dorpat*, edited by Professor Unverricht, 1893.

It is almost impossible to pump the stomach completely empty. Ost always performed his experiments in the morning, when he could be reasonably certain that the stomach was empty. It was necessary to determine to what extent the method was able to yield useful figures in regard to the capacity of the stomach, and it was found that in those cases where a number of experiments were performed in one day a certain definite quantity of air could be pumped out at first. In later experiments this quantity increased, and in still later ones decreased again. This seems to indicate that the stomach gradually acquires a certain tolerance, but that toward the end of the experiments a certain amount of air probably escapes from the stomach through the pylorus. In healthy organs the results fluctuate within wide boundaries. Ost, therefore, decided that the amount of air that can be pumped into the stomach is no criterion for the capacity of the organ. At all events, Ost's experiments, the details of which we cannot describe, show that it is impossible, even approximately, to determine the capacity of the stomach by measuring the amount of air pumped into it. One of the chief reasons for this, according to Ost, is the fact that much of the air escapes into the intestine. For this reason the procedure described is not a practical one.

(*j*) **The Method of Dehio.**[1]—This method purposes determining the distention of the stomach; at the same time, the degree of mechanical sufficiency or insufficiency.

If a healthy person drinks a quarter of a liter of water that is not too cold, an area of dulness can be percussed in the angle between the liver and the lung, or the liver and the heart, when the subject is in the erect position; upward and to the right this dulness merges into the heart- and liver-dulness; upward and to the left it is clearly distinguishable from the loud sound of the left lung; and downward it is clearly distinguished from the tympanitic or loud sound over the rest of the abdomen. If the subject drinks a second glass of water, this dulness extends further downward and laterally; after the third or the fourth glass this dulness becomes still greater. It will be found that a half pint of water will bring the lower boundary of the stomach-dulness in the median line to about 4½ inches below the lower end of the corpus sterni. If the stomach is filled with more fluid, the lower boundary will gradually drop. After the second glass it is usually 1 inch lower than after the first. The third glass will force it downward another inch, and the fourth glass, 1 inch further. In an overwhelming number of cases it has been found that the lower boundary of the stomach remains several centimeters above the umbilicus, and is only rarely at a level with the umbilicus after one quart of fluid. Dehio, as well as Penzoldt and Weil, has never found this boundary lower in normal subjects.

If the patient is now instructed to lie on his back, tympanitic sound will naturally appear where dulness was before. This change from dulness to tympanitic sound with a change in the position of the patient will demonstrate conclusively that the area corresponded to the stomach; we are enabled to draw certain conclusions in regard to the size and extent of this organ from the extent of the dulness after drinking different amounts of water.

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[1] *Verhandl. d. VII. Cong. f. innere Med.*, 1888.

In very obese subjects this method may fail because no distinct differences in the percussion-sounds can be elicited through the thick abdominal walls. If the colon is distended with fecal masses, the results of percussion may be obscure. All that is needed in this case is to empty the colon.

Similar results as from drinking water may be obtained if the subject first is examined when the stomach is empty, and later again, half an hour after an ordinary midday meal.

The method of Dehio, which we have just described, is essentially different from the ordinary methods of percussing the stomach. As a rule, the stomach is percussed whenever and wherever it is convenient. In Dehio's method percussion is more methodic, inasmuch as the stomach is at first examined when empty, and later again step by step as the dulness of the stomach extends, corresponding to the different amounts of fluid ingested or corresponding to the position of the patient. One of the chief advantages of this method is the ability to obtain a fairly clear picture of the changes in the form, position, and size of the stomach when the organ is filled to different degrees.

This method, therefore, does not only yield topographic information, but at the same time determines to a certain extent the powers of distention of the organ.

In diseases of the stomach the results obtained are somewhat different. Dehio was enabled to determine in the majority of cases of chronic dyspepsia with pathologic weakness of the muscularis and atony of the stomach-wall that the dulness caused by filling the stomach with water extended further down than normal. One glass of water occasionally caused the dulness to extend downward to a point corresponding to the boundary of the stomach after the ingestion of two glasses in normal subjects. Sometimes two glasses of water would cause the dulness to extend to the umbilicus or still further down. Occasionally it was found that a single glass of water forced the boundary to the umbilicus.

If this occurs, Dehio feels justified in concluding that the stomach-wall has lost some of its normal contractility, and that the cases are either sufferers from atony or insufficiency of the stomach.

If there is pronounced gastric ectasy, so that the stomach is permanently in a condition of abnormal flaccidity and distention, a single glass of water will frequently cause dulness to appear below the navel, even as far down as the crest of the ilia, whereas in simple insufficiency this will never occur.

Percussion of the stomach with progressive filling of the organ with water enables us, therefore, to gain much information in regard to the mechanics of the organ, its elasticity, and its powers of distention.

The results obtained are reliable. The passage of the stomach-tube is not necessary, and the method is easy of execution. I think, therefore, that I can justly recommend it for general practice.

In two points, however, I must differ from Dehio. It is true that lowering of the stomach boundary after the ingestion of relatively small quantities of water demonstrates that the organ can be readily stretched —in other words, that its elasticity is diminished. At the same time it does not, as Dehio concludes, prove that the motor power is reduced.

In other words, abnormal distention of the stomach does not, *per se*, indicate a diminution in the motor powers of the organ. The resistance offered to distention need not equal the contractile powers of the musculature. For this reason the method of Dehio cannot be employed for determining the motor powers of the stomach.

The second point in which I differ from Dehio is the following: He claims that his method is far preferable to inflation with carbonic acid gas or insufflation with air. I do not agree with him when he says that inflation with carbonic acid gas only teaches us that the stomach can be abnormally distended, but does not show whether this abnormal distention is due to a weakness and insufficiency of its musculature or is a permanent state—dilatation of the stomach proper; nor when he says that distention of the stomach with carbonic acid gas produces the same picture in insufficiency of the stomach as in permanent ectasy.

I am of the opinion that inflation with carbonic acid gas and insufflation with air should not be compared with Dehio's method. Dehio's method is no more suited to determine the motor power of the stomach than are inflation with CO_2 and insufflation of air. All three methods enable us to measure the powers of distention of the stomach, and, to a certain degree, the size and position of the organ; inflation and insufflation of air, however, give us a more complete picture of the form and position of the stomach than Dehio's method of water-drinking. The methods supplement each other; both have their advantages and both are limited in certain directions. However valuable I may consider Dehio's method, I cannot concede that it renders inflation with carbonic acid gas and insufflation with air superfluous. [Furbringer[1] advises inflation by introducing the tube only to a point about the middle of the esophagus, and pumping in air through the tube in this position. He finds that it incommodes the patient but slightly and prevents retching.—Ed.]

Auscultation of the Stomach.—This procedure is of subordinate importance. Different sounds are distinct, as follows: first, sounds that are produced in the esophagus by the act of deglutition; second, sounds that originate in the stomach. The former, the so-called deglutition sounds, are of no value in the diagnosis of diseases of the stomach.

If the ear is applied to the region of the ensiform cartilage or to the left side of the spinal column in the region of the ninth or the tenth rib, two sounds will be heard on deglutition: the first appears almost immediately after swallowing, and resembles the sound produced by squirting fluid through a tube containing air. Meltzer, therefore, called it "*Durchspritzgeräusch*"; Ewald called it the primary sound. A few seconds later (usually six to twelve) a second sound is heard that is more rattling in character, like a coarse râle. Ewald calls this a secondary sound; Meltzer, a "*Durchpressgeräusch.*" The first sound is not constant; the second one more so, but may also be absent. Meltzer assumes that the first sound is caused by relaxation of the cardia, but Ewald combats this view. The only diagnostic point is the absence of this secondary sound in more or less complete occlusion of the cardia [or spasm of the same].

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[1] *Boas' Arch.*, vol. vi., pt. i.

In order to be of diagnostic value, this sound must be constantly absent; it is occasionally absent in normal subjects. If the cardia is narrowed, the second sound will be delayed, and, according to Boas, may not be heard for fifty to seventy seconds.

Sounds that originate in the stomach itself are more important. They are the so-called splashing or succussion sounds and the gurgling or "Klatsch" sounds. I need not speak extensively of these sounds in this place, as I have already mentioned them in the discussion on Palpation of the Stomach. In order that splashing should be heard, air and liquid must be present.

The sound is best produced when the patient is lying on his back, provided, of course, the walls of the abdomen and the stomach are not under too great tension. Occasionally this sound is heard spontaneously when the patient turns over or performs some violent movement. Some patients are able to elicit it by voluntary contraction and relaxation of the abdominal walls. Diagnostically, it is relatively insignificant. Even if it were demonstrated conclusively that the sound originates in the stomach, it would be pathologically significant only if it occurred at a time when the stomach should normally be empty—for instance, early in the morning—or if it were heard in a location outside of the boundaries of the normal stomach. Splashing can always be stopped if the stomach is pumped out; if after this the sound is produced, it did not originate in the stomach, but in the intestine, probably the colon, for it can be caused here under identically the same conditions as in the stomach. It must be remembered, of course, that in certain cases of ectasy of high degree it is impossible or at least very difficult completely to evacuate the stomach.

Gurgling and "Klatsch" sounds are heard if the stomach contains only air, so that these are noted most frequently when the stomach is empty. Many people are able to cause sounds of this character by contracting and relaxing the abdominal walls, and patients are apt to attach a great deal of importance to this power. In reality, these sounds are of subordinate diagnostic significance.

Sounds that may be called broiling or bubbling sounds are occasionally heard over the stomach. They may be heard in normal cases during digestion, and they are pathologic if they are particularly loud or appear at a time when the stomach should be empty. I have heard them with particular frequency in cases of hypersecretion and pronounced ectasy in which there was much development of gas. They may also be heard when the stomach is being inflated with carbonic acid gas.

Occasionally the heart-sounds, modified so as to give a metallic resonance, are heard over the stomach if the organ is filled with air and distended. Riess[1] believes that a peculiar metallic heart-sound heard over the stomach is a rare sign of pericardial adhesions, and states that the occurrence of this sound in this region is favored by the intimate connection existing between the wall of the heart and of the stomach. At the same time metallic heart-sounds are heard under so many different conditions that no definite conclusions can be drawn from either occurrence. The fact that they are transmitted to the region of the stomach is, of course, of subordinate importance.

Gabbi[2] has shown that respiratory sounds, particularly if breathing is enforced, can be heard over the stomach region, especially where adhesions exist between the two layers of the pleura, chiefly between the base of the left lung and the diaphragm. In chronic peritonitis and fluid exudation into the abdominal cavity respiratory sounds are heard over the whole abdomen. [By the conjoint methods of auscultation and percussion or palpation some clinicians are able to reach a fairly correct idea of the boundaries of the stomach. A binaural stethoscope with a small bell is applied, and, while palpation or percussion is practised, the instrument is moved about from point to point. The character of the sounds produced by the intragastric splash is sometimes sufficiently characteristic to enable one to make satisfactory localization. A friction-sound may also occasionally be heard in case of perigastritis by practising auscultation.—Ed.]

Finally, a peculiar sound like the report of a gun has been heard by different investigators in cases of rupture of the stomach.

The Use of the Stomach-tube for Diagnostic Purposes.—All the methods described so far aim at determining the size, the form, the position of the stomach, the presence or absence of pain or of tumors. We can learn from them whether the stomach is large or dilated, whether it is dislocated, whether it is painful, and in which region, whether tumors are present and where they are situated, whether they are movable or fixed, etc. All this information is, of course, important, and in a great many cases these methods, combined with the anamnesis and the general health of the patient, may enable us to make a correct diagnosis. In many, in fact in the majority, of cases these methods of examination are sufficient.

The ideal object would be to see the interior of the stomach and to examine its mucous lining, but whatever information simplified methods of gastroscopy may give us in the future, even this method would not be sufficient in all cases. The practising physician will never be content with an anatomic diagnosis nor with determining the name of the disease. What he wants is a complete insight and understanding of the perversion of physiologic function so that he may be able to compensate any deficiency of function. The physician wants to know more than that a tumor is present in this or that location, or that the mucous lining of the stomach is inflamed in this or that area, or is ulcerated. He wants to know to what extent the specific functions of the stomach are perverted.

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[1] *Berlin. klin. Wochenschr.*, 1878, No. 51, and *Zeitschr. f. klin. Med.*, vol. xvi.

[2] *Riv. clin.—Arch. ital. di clin. med.*, 1889; *Centralbl. f. klin. Med.*, 1890, No. 41.

An exact knowledge of these disturbances is necessary before a rational therapeusis can be instituted. In many cases where a direct cure is not possible, an indirect one may be brought about by removing and compensating these disturbances of function. The stomach-sound or stomach-tube is the instrument that gives us information in regard to perversions of function.

It would lead us too far to recount the history of the stomach-tube, even briefly. I refer those who are particularly interested in this subject to the dissertations by Leube[1] and Sticker.[2]

Kussmaul,[3] in 1867, demonstrated the therapeutic value of the stomach-sound in a number of cases of dilatation of the stomach. Before his day a few isolated instruments were described, but he was the first to recognize the significance of this procedure. The employment of the stomach-sound has slowly but steadily increased, so that to-day it is considered one of our most valuable therapeutic adjuvants. Leube,[4] in 1871,—that is, a few years after Kussmaul,—used the stomach-sound for diagnostic purposes. The reason why this instrument was not used as a diagnostic adjuvant until the second half of the eighties was due to the fact that in the beginning the results were unsatisfactory, and that the difficulties of using the apparatus were considerable. Even twelve years after Leube's[5] recommendation the results were very unsatisfactory, and this may explain why this important method was not universally adopted for so long a time.

Leube purposed determining, on the one side, the time of digestion, and, on the other, the strength of gastric secretion. I will refer to his method of determining the time of digestion later. In this place we are concerned chiefly with the strength of gastric secretion. The reason why Leube's experiments in this direction were so unsatisfactory was that he operated on an empty stomach. Leube believed that if the examination was made during digestion,—that is, at a time when the food was being changed in the stomach,—a number of difficulties would be presented, as follows: The quantity of gastric juice that would be present at any given moment and was actively concerned in digestion would not be the same at all times; parts of it would be absorbed by the food; the secretion would be influenced by the mechanical and chemical consistency of the food, that was certainly never uniform, and, consequently, the determination of pepsin and hydrochloric acid, particularly the quantitative ones, would be inconstant.

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[1] Leube, *Die Magensonde: Die Geschichte ihrer Entstehung und ihre Bedeutung in diagnostischer und therapeutischer Hinsicht*, Erlangen, 1879.

[2] Sticker, "Magensonde und Magenpumpe," rep. from *Deutsch. med. Zeit.*, 1887, No. 74, etc.

[3] See *Berichte d. 41. Versamml. deutsch. Naturforscher u. Aerzte*, 1867, and *Deutsch. Arch. f. klin. Med.*, vol. vi.

[4] *Bericht d. Rostocker Naturforscherversammł.*, 1871.

[5] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii.

Acting on these scruples, Leube decided to analyze the gastric secretion of the empty stomach instead of the full one. He employed mechanical, chemical, and thermic irritation in order to stimulate the secretion of gastric juice, and ultimately arrived at the conclusion that thermic irritation with iced water was the most suitable one. He found, however, that even in healthy subjects this irritant was frequently insufficient to cause a secretion of gastric juice. In severe cases of dyspepsia that were not purely nervous in type, chemical or thermic irritants frequently caused the secretion of a fluid that contained no acid and no pepsin. In 1884 I[1] performed a number of control experiments that showed that Leube's ice-water method frequently causes no excretion of gastric juice, and that the secretion pumped out was never so active as that obtained if the stomach was evacuated at the height of digestion. At that time, therefore, I recommended examining the secretory activity of the stomach, not when the organ was empty, but at the height of digestion. This method is universally employed to this day.

The sound may be used for the diagnosis of a variety of conditions, namely:

1. In order to determine whether the esophagus and the cardia are open.
2. In order to determine the size of the stomach or the location of its lower boundary (see the section on Leube's Method).
3. In order to determine the time of digestion.
4. In order to remove stomach-contents for the purpose of examining the chemism of digestion.

The last two objects may frequently be attained at the same time. If the stomach is pumped out a certain time after the digestion of a test-meal, conclusions may be drawn from the quantity of residue obtained as regards the duration of digestion, the motor power of the stomach, and the chemical transformations of the food. In this way some idea of the secretory activity of the stomach may be obtained.

The Stomach-tube.—The word stomach-sound is frequently used in place of the correct name, stomach-tube. Ewald is correct when he says that the term "sound" is not an appropriate one when we consider the uses to which the instrument is put nowadays. A sound means an instrument with which we feel or sound, and our stomach-tubes are made of such soft and elastic material that they certainly are not fitted for sounding. All this is true; at the same time, the word sound is so universally introduced that it will be hard to replace it. Of course, there is nothing in a name.

Formerly, stiff sounds were used. In 1870 Jürgensen[2] employed a soft-rubber tube for pumping out the stomach. His instrument ended in an ivory knob that was perforated. In order to introduce this tube, he used a wire. I used this instrument immediately after it was described, and discovered soon that it could be introduced very well without a wire. Others had the same experience. In 1875 Ewald called attention to the fact that any piece of rubber-tubing that has sufficient resistance to allow its introduction into the stomach without a guide would answer the purpose. Many physicians will have discovered this independently, particularly whenever it was necessary to wash out the stomach suddenly, as in cases of poisoning when a stomach-tube was not available. Under these circumstances everybody will pick up and use any piece of rubber-tubing that happens to be convenient.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxvi.

[2] *Ibid.*, vol. vii.

Nowadays, soft tubes are almost universally employed. Nélaton's urethral catheters are the most frequently employed. This instrument is about 75 cm. (29½ inches) long, and may be of different lumen—on an average, about 6–7 mm. (¼ inch).

I am in the habit of using and having ready a number of stomach-tubes. The lower end should be dull or conical. The majority of stomach-tubes have two lateral openings or windows at the lower end. The lowest portion of the tube is usually closed (see Fig. 5). An instrument is not well constructed in which there is a long closed piece below the lowest window, because particles of food may accumulate in this cul-de-sac and render it difficult to keep the instrument clean.

In order to do away with this drawback Ewald[1] recommends tubes that are open below; instead of having one large window, they have a number of small holes on the side which are a little larger than pin-heads (Schütz) (see Fig. 6).

Of late years Rosenheim[2] has recommended a similar modification of the stomach-tube which he calls a stomach-douche. Rosenheim did not recommend this modification for diagnosis, but only for irrigation of the stomach mucosa with medicated fluids. His instrument, therefore, is perforated by a large number of small openings, and has a hole at the end some 3 to 4 mm. (⅛ inch) in diameter.

Personally, I am in the habit of using tubes with two large lateral openings and a blind end (Fig. 5). This I use for diagnosis. For therapeutic purposes I use tubes with many small openings. I do not think that the small openings are advantageous when it is desired to pump out the stomach.

It is important, of course, to clean and disinfect the tubes after they have been used. In private practice the patient should have his own tube. Tubes that are used in cancer or syphilis should not be used for other patients and should be marked; the same applies to tuberculosis. Ordinary disinfectants can be used to sterilize the sound. Kuttner[3] has advised a special apparatus for sterilizing soft catheters, and Boas recommends it. In this apparatus the instruments are disinfected with streaming steam. In large institutions in which a certain number of stomach-cases are examined with the sound every day an apparatus of this kind is probably useful. Personally, I have gotten along very well without one.

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[1] *Klinik d. Verdauungskrankheiten*, ii., third edition, p. 7; *Berlin. klin. Wochenschr.*, 1875, No. 1.

[2] *Therapeut. Monatsh.*, 1892, p. 382.

[3] *Ibid.*, 1892.

In order to procure some of the stomach-contents the sound must be connected with a rubber tube and a glass funnel. The external end of the stomach-tube is connected with a short glass tube of from 7 to 10 cm. (3 to 4 in.), and this, again, is attached to a long, soft-rubber tube that leads to a glass funnel. This arrangement is simple and sufficient for all purposes.

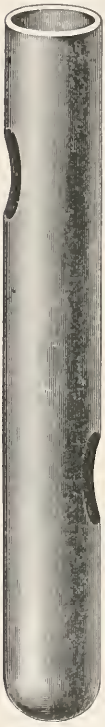

FIG. 5.

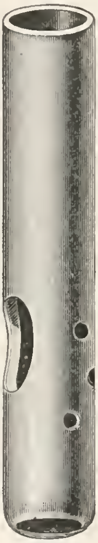

FIG. 6.

A stomach-pump is superfluous and should not be used. The same applies to hard stomach-tubes. Several cases are on record in which aspiration by pumping has torn off small pieces of stomach mucosa. Leube,[1] Wiesner,[2] Ziemssen,[3] Schliep,[4] Huber,[5] Hänisch,[6] and others have reported such accidents. Undoubtedly such mishaps have occurred more frequently than they have been reported. I myself have seen numerous accidents of this kind when I was still in the habit of using the pump. Crämer[7] has reported 2 cases, and, quite recently, Ebstein[8] a third one.

Crämer's cases are ambiguous because he himself states that the edges of the openings of his sound were not quite smooth. Personally, I have seen the tearing-off of pieces of mucous membrane even when the stomach-tube was carefully used. Boas is right when he states that in the most careful expression of the stomach-contents, particularly in chronic catarrh, exfoliation of small pieces of mucous membrane may occur. Boas observed the same in cases of great hyperacidity. Bad results were never seen. In general, the introduction of a soft stomach-tube and expression without the pump may be characterized as manipulations that are altogether without danger. I have personally performed this operation in many thousands of cases, and have never seen untoward results from aspirating the stomach for diagnostic purposes. For sixteen years we followed the practice in all cases of stomach-diseases in which some contraindication was not discovered. Occasionally there is very violent gagging, so that the sound and some food

[1] *Deutsch. Arch. f. klin. Med.*, vol. xviii.

[2] *Berlin. klin. Wochenschr.*, 1870, No. 1.

[3] *Deutsch. Arch. f. klin. Med.*, vol. x

[4] *Ibid.*, vol. xiii

[5] *Ibid.*, vol. xxi.

[6] *Ibid.*, vol. xxiii.

[7] *Münch. med. Wochenschr.*, 1891, No. 52.

[8] *Berlin. klin. Wochenschr.*, 1895, No. 4.

are vomited. This is, of course, disagreeable; we have never, however, seen symptoms of suffocation or so-called aspiration-pneumonia. Patients, it is true, frequently complain that they cannot breathe when the sound is being introduced; they think that they are suffocating. This sensation stops at once, however, if they are instructed to breathe deeply instead of holding the breath, as they so frequently do when the sound is introduced. Gagging has nothing whatever to do with the introduction of the sound into the stomach, but is caused by irritation of the pharynx. As soon as the patients become accustomed to this irritation, the gagging stops. In patients who are particularly sensitive the mucous lining of the pharynx may be painted with cocain. I have found that the latter procedure is only rarely necessary, and only in the first few attempts to pass the sound. Hyperesthesia of the mucous membrane of the pharynx is found more frequently in men than in women. This is probably due to the fact that catarrh of these parts is seen more frequently in the former. Artificial teeth should be removed before the stomach-tube is introduced.

Technic of Introducing the Stomach-tube.—The introduction of the stomach-tube is a very simple manipulation. If the patient is not afraid, he may be allowed to pass the tube himself. It is not well to oil the sound or to lubricate it with fat or vaselin, although many do this; it is better to dip it into warm water. The patient should be placed opposite the physician, and should be instructed to take hold of the moist tube himself and to place it in his mouth. As soon as the tube reaches the base of the tongue the patient should be instructed to swallow. When this is done, the tube enters the pharyngolaryngeal sinus alongside of the root of the tongue, just as a morsel of food would. In this way the tube is engaged in the first part of the esophagus. The patient is instructed to breathe quietly and regularly, and the tube is then pushed slowly but rhythmically downward until the stomach is reached.

If the patient is afraid to introduce the tube himself, the physician should push it beyond the tongue with his right hand, and as soon as the tube reaches the root of the tongue, the patient should be instructed to swallow. This will cause the sound to glide into the first portion of the esophagus. After this the physician proceeds as above. I wish to advise against depressing the tongue with one or several fingers of the left hand while the right is introducing the tube. This procedure is disagreeable to the patient, and not only does not facilitate the introduction of the tube, but renders it more difficult. The patients will involuntarily hold their breath. Besides, the introduction of the fingers into the mouth will cause vomiting in many people, and in this manner render the downward movement of the tube more difficult.

If the tube is manipulated correctly, there is no danger of entering the larynx instead of the esophagus. Beginners may sometimes fear that this has happened when they see the patient turn cyanotic, but this symptom is due only to the fact that the patients hold their breath. Many patients imagine, particularly when the stomach-tube is introduced for the first time, that they cannot breathe, and despite all encouragement on the part of the physician they involuntarily hold their breath. As a rule, an energetic command to breathe deeply and regularly will lead to success.

I wish to mention one particular danger, and that is the following: The tube may slip down into the stomach. This accident, of course, is rare. Leube[1] reports a case of this kind in which the patient swallowed the whole tube. The subject was a woman of about sixty with pronounced gastrectasy, whose stomach was washed out every day for many weeks. She was dismissed with the instruction to have her stomach washed out for some time longer by her physician. The patient, however, did not employ a physician, but a barber's assistant, who aided her in the lavage. On one occasion the connection between the sound and the tube to which the funnel filled with water was attached became loosened. The water gushing from the funnel forced the sound into the mouth. The patient was startled and made swallowing movements, and the sound disappeared. A physician was called in and attempted in different ways to extract the sound, but failed. The patient then returned to Leube. The sound could be felt very distinctly through the abdominal walls. All attempts at extracting it failed, nor did vomiting caused by apomorphin lead to anything. On the contrary, fever occurred, probably as a result of slight injuries that had been inflicted on the esophagus by the pointed instruments that were employed at the patient's home in attempting to extract the sound. The fever became quite high, so that the patient was alarmed and returned home without having the tube removed. Her condition grew worse, but later the fever decreased. On the ninth day after the catastrophe there were sudden nausea and a desire to cough, combined with a feeling of suffocation. The patient pushed her fingers down into the pharynx, found the sound behind the tongue, and pulled it out with one jerk.

[It would seem unnecessary to call attention to the dangers of using a cracked or otherwise weakened stomach-tube; and yet, the interesting case reported by Friedenwald[2] points out the danger of carelessness in this respect. The case was that of a colored woman, aged twenty, upon whom a quack doctor passed a defective stomach-tube, and in attempting to remove it left a portion behind. No evidence of the presence of the tube could be felt upon palpation through the abdominal wall. Upon entering the Providence Hospital, Baltimore, Dr. Harris performed a gastrotomy and removed two pieces of stomach-tube, one 9¾ inches and the other 6¼ inches in length. The patient made an uninterrupted recovery.—Ed.]

Since reading about this case of Leube's I have always instructed the patient to hold the sound tightly between the fingers at that end which is immediately in front of the glass tube.

[All this embarrassment may be avoided by having the two sections of the tube united with a metal clamp, as found in the tube employed with the gastric electrode by Stockton.—Ed.]

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii., p. 6.

[2] *American Medicine*, August 2, 1902.

Indications for Using the Stomach-tube.—The question when and when not to use the sound for diagnostic purposes has been answered in different ways. In my opinion the question is a simple one. The sound should be used wherever diagnostic or therapeutic advantages can be expected from its employment. It may be contraindicated by the nature of the disease or by the general condition of the patient. I do not agree with those authors who advise not to use the sound whenever the nature of the disease can be diagnosed by other methods. There is no doubt that certain cases of carcinoma, of neuroses, or of ulcer of the stomach can be diagnosed without sounding. In fact, older physicians had to get along without this adjuvant. At the same time I consider sounding indicated in these cases unless there is a direct contraindication. Every physician of experience has observed cases in which he was positive of his diagnosis, but afterward found that he was mistaken. If for no other purpose, everything should be done to render the diagnosis absolutely positive as long as the patient's condition is not aggravated or his life endangered by such procedures. There is another factor, however; there are, it is true, cases in which the diagnosis can be made without sounding, and many authors, as Boas, consider the exploratory employment of the sound contraindicated in cases where the diagnosis can be made from other symptoms. I do not agree with them. I am not content to know that the patient is suffering from cancer, nor is it immaterial to know whether his motor power is good or bad, whether his digestion is fair, whether the secretion of gastric juice is stopped. All these questions can be answered only by sounding. The prognosis and treatment will vary according to the results obtained. Sounding, of course, is contraindicated if a gastric hemorrhage has recently occurred or if there is peritonitis. It is also counterindicated in cases where the patient is very weak, where there is high fever, where the patient is near unto death, in the last months of pregnancy, in the presence of an aneurysm of the aorta, etc. It is, of course, impossible to enumerate all the contraindications. Every physician will have to draw his own boundaries and will have to determine for himself where sounding is necessary and where it is not permissible—in other words, where sounding cannot be performed without damaging the patient and where it is dangerous.

Methods for Obtaining Stomach-contents.—Different methods have been described for obtaining stomach-contents. As soon as the sound has entered the stomach, the next task is to obtain some of the contents of the organ.

There are two methods for removing stomach-contents through the sound: The one is aspiration through an aspirator of some kind that is attached to the end of the tube; the other is the so-called method of expression of Boas and Ewald. Here the patient himself exercises pressure and forces the contents of the stomach into the sound.

The first method by suction or aspiration can be performed with the aid of a stomach-pump. Kussmaul did this in his first experiments that he performed for therapeutic purposes. A disadvantage of this method is that a rather complicated apparatus is needed, and that injuries are more liable to be inflicted on the mucous lining of the stomach.

Instead of using the stomach-pump, bottle aspirators, constructed after the model of Potain's apparatus for the evacuation of pleuritic exudates, may be employed. Different modifications of this apparatus have been constructed. Any one can put such an apparatus together as follows: Into a bottle is placed a cork with two holes, through which pass two glass tubes; one of these is connected with the stomach-tube, the other with a Potain syringe, or a balloon aspirator may be used (Ewald[1] and Boas[2]). Ewald connects the stomach-tube with a Politzer bag by means of a tube of hard rubber or of bone. The opening in the Politzer bag should be rather large—about as large as a little finger. If the bag is compressed before connecting it with the stomach-tube, and is then allowed to expand after the connection has been established, the stomach-contents can be sucked into the rubber bag. Ewald recommends using a bag of some 250 c.c. (8½ fluidounces), made of good strong rubber, and having an upper orifice of at least 15 mm. (⅝ inch) in diameter.

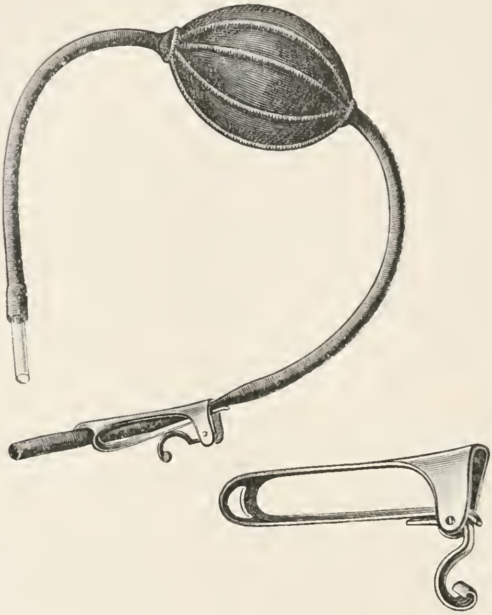

Fig. 7.—Aspirator of Boas, one-half natural size.

Boas objects to this method on the ground that it is too tedious, and because the rubber bag must be removed after each aspiration.

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[1] *Loc. cit.*, p. 13.

[2] *Loc. cit.*, p. 128.

Boas used another form of aspirator that is constructed on similar principles It consists of a rubber ball, to which are attached two long rubber tubes (see Fig. 7). This bag is connected with a stomach-tube by a piece of rubber tubing. There is a cock on the tube that is not connected with the stomach-sound ; when this cock is opened and the bag is compressed, the air is removed from it ; if the cock is closed, the bag expands and aspirates the stomach-contents ; if the cock is opened again, the contents of the bag may be expressed into some vessel if that piece of the tube that is connected with the stomach-sound is compressed. By alternately sucking and pressing, the stomach-contents may be removed. Jaworski and others have described similar but more complicated apparatus.

If I may be allowed to express my opinion in regard to these apparatus in this place, I would say that however useful they may be, we can still get along very well without them. I do not wish to depreciate the value of these apparatus, nor rob those that have invented them of the credit they are entitled to, but personally I have always employed the simplest and most natural methods for obtaining stomach-contents for diagnostic purposes—namely, expression. This method has been described by a number of investigators, but Ewald and Boas[1] are the first who used it in a rational and systematic manner and caused its general adoption. Ewald and Boas, in 1885, showed that it is an easy matter to express the stomach-contents by simple contraction of the abdominal muscles, and that all that is necessary to force stomach-contents into the tube and the funnel is contraction of these muscles.

Ever since 1879, the time at which I began my examination of the stomach-contents for diagnostic purposes, I have allowed the patient to express the contents of the stomach, and have in rare cases only used the pump. Many patients involuntarily force the stomach-contents into the tube as soon as the sound is introduced ; others gag, so that it is frequently an easy matter to start these reflex movements by moving the sound up and down. I assume that everybody who has examined many stomachs has had a similar experience. Ewald and Boas at the same time deserve full credit for having employed this method systematically, and for having described it exhaustively.

It is hardly necessary to call attention to the fact that it is necessary to lower the funnel in order to allow the stomach-contents to flow out. Occasionally it will be found that no stomach-contents enters the sound or the funnel ; this may occur in case the openings in the lower end of the sound become occluded, or if the stomach-contents is too coarse to pass through these openings, or, finally, if the tube has been inserted too far and has become bent within the stomach so that the opening at the lowest end does not dip into the fluid within the organ. In the latter case all that is needed is to pull the sound out a little. If the openings are stopped up with thick morsels of stomach-contents, and if none of the aspiration apparatus is handy, the tube must be removed, cleaned, and reintroduced.

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[1] *Virchow's Arch.*, vol. ci., p. 330.

Friedlieb[1] has devised an apparatus that is very practical in such an emergency. It is a simple instrument, and can be attached to an ordinary stomach-tube; it consists of an egg-shaped rubber bag about as large as a goose-egg (see Fig. 8). In either end is inserted a glass tube. This instrument is inserted into the stomach-tube in place of the glass tube that connects the rubber tube and the stomach-tube. The method of using this apparatus is self-evident.

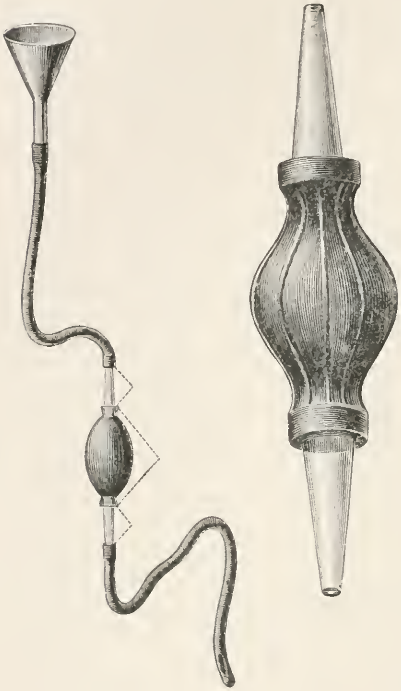

Fig. 8.—Friedlieb's apparatus.

My former assistant, Dr. Strauss,[2] has devised another apparatus that is more useful and more reliable, although it seems to be more complicated on first sight. I have employed this apparatus repeatedly of late and have found it useful. It is constructed as follows (see Fig. 9): In place of the ordinary glass tube that usually connects the funnel-tube and the stomach-tube a T-tube is inserted, the vertical branch of which is connected with a double bellows. All the tubes that leave this T-tube are connected with clamps that can be opened or closed as desired. Any form of rubber-tube clamp can be used, but the one shown in the illustration seems to be the most practical one. The position of the different clamps can be seen from Fig. 9. The whole apparatus is constructed like all others that are used for washing out the stomach if clamps *t* (funnel-tube) and *m* (stomach-tube) are open, and clamp *b* (bellows) is closed. If it is desired to remove a morsel of stomach-contents that occludes the stomach-tube, clamp *t* is closed, *b* and *m* are opened, and air is pumped from the bellows. If *m* is closed and *b* and *t* are opened, the contents of the funnel may be forced out by expression. In the figure *b* and *m* are open, while *t* is closed.

One of the chief advantages of this apparatus is that we can aspirate stomach-contents, can wash out the stomach, and can inflate it for diagnostic purposes without changing the apparatus. It also enables us to remove any obstruction in the stomach-tube more simply than this can be done with any other form of apparatus. At the same time this apparatus, like all others, suffers from disadvantages inherent to the aspiration of stomach-contents in general.

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[1] *Deutsch. med. Wochenschr.*, 1893, No. 51.

[2] *Therapeut. Monatsh.*, 1895, No. 3.

While I can see some advantage in using any one of the methods just described, I cannot see that the following method described by Gross is of any value.

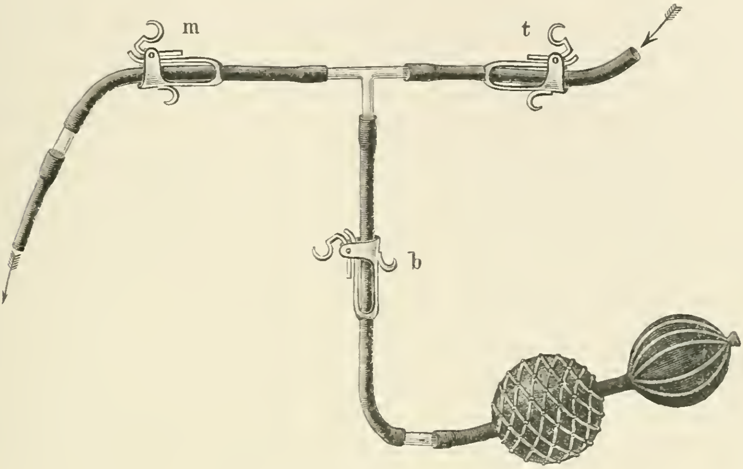

FIG. 9.—Apparatus of Strauss.

Gross [1] claims that the disagreeable features of introducing the sound are reduced to a minimum by his method.

The apparatus (see Fig. 10) consists of three parts—namely, a stomach piece, a middle piece, and an end piece. The stomach piece (*a*) is a long, thin, Nélaton catheter, to the end of which an oval-shaped piece of hard rubber is attached. The middle piece (*b*) consists of a concentrically constructed glass ball in which the stomach-contents is collected; attached to this glass ball by a short rubber tube is a manometer for determining the pressure in the tube. The end piece (*e*) terminates in a modified Politzer bag with a valve or in a rubber ball with a clamp, or in a mouth-piece into which the physician is expected to blow.

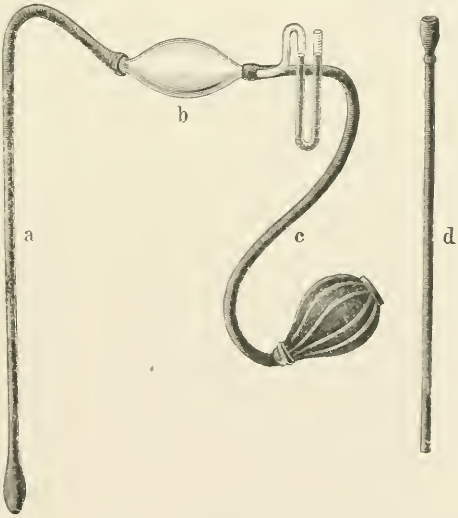

FIG. 10.—Apparatus of Gross.

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[1] *Fortschritte d. Krankenpflege*, December, 1893; *Therapeut Monatsh.*, 1894, No. 12.

I cannot see that this apparatus has any particular advantages, for if Gross chooses to employ a stomach-tube that is as thin as a Nélaton catheter and resembles those that we use for washing out the stomach in children, he naturally presupposes that the stomach-contents will be altogether fluid. If larger pieces are present in the stomach, the sound will be occluded. Aspiration is employed in this method and certainly presents no new features.

It may occasionally happen that nothing will be aspirated in an attempt to obtain stomach-contents for diagnostic purposes, not because the sound is occluded or bent, but because the stomach is empty, owing to the fact that the time chosen for aspirating the stomach-contents was too late. That this is the case can readily be determined by allowing water to flow into the stomach and siphoning it out again. In a case of this kind the experiment will have to be repeated on the following day, but at an earlier hour. It is not well to limit the examination to one attempt; particularly if it is desired to study the chemistry of the stomach-contents, aspiration should be repeated a number of times. Aside from the fact that the first examination may fail either because the tube becomes occluded or because the examination is made too late, it may happen that residual food is present in the stomach, which may influence the results obtained from the chemical examination of the test-meal. In other words, in order to obtain positive and reliable results, examination of the stomach-contents should be repeated a number of times.

For completeness' sake I will describe a few methods that were originally devised for the purpose of doing away with the introduction of the sound and limiting the examination to small quantities of stomach-contents.

Edinger[1] was the first to report such a method from my clinic in 1881. Small pieces of sponge that are neutral in reaction are attached to a silk thread and inclosed in very small capsules of gelatin; the thread passes through the capsule. The capsule is swallowed, and the sponge pulled out after fifteen minutes, its contents expressed, and a number of color-reactions performed with the drop or two of gastric juice that is obtained in this way.

I never liked this method. First of all, a good deal of the contents of the sponge is lost in pulling it out, and, besides, a certain amount of mucus, which neutralizes the gastric juice, is absorbed; the method is just as disagreeable to the patient as the introduction of an elastic stomach-tube. A thread running across the tongue and dangling from the mouth is just as uncomfortable as the sound; besides, the latter instrument is inserted for a few minutes only, whereas the thread and the sponge must remain in place for a quarter of an hour. The amount of gastric juice obtained is never sufficient for quantitative determinations, and is of value only for certain qualitative tests.

The same drawbacks are attached to the method devised by Späth.[2]

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[1] *Deutsch. Arch. f. klin. Med.*, 1881, vol. xxviii.

[2] *Munch. med. Wochenschr.*, 1887, No. 41.

Späth employs small balls of elderpith that are attached to a silk thread and are saturated with Congo-red. If the stomach contains hydrochloric acid, these little balls are colored blue. If nothing more is desired than to elicit the Congo reaction, it is a much simpler method to introduce the sound rapidly, to pull it out again, and to examine the fluid found in the lower opening of the sound with Congo-paper. Einhorn[1] has described another method for obtaining stomach-contents. He allows the patient to swallow a small, egg-shaped apparatus that is attached to a thread. The original apparatus of Einhorn (see Fig. 11, *a* and *b*) consists of two hemispheres that are fitted into each other in such a manner that the outer hemisphere is larger, but has a smaller opening than the other one. The thread is attached to the latter. As long as the apparatus is dangling from the thread the smaller hemisphere closes the opening in the larger one (see Fig. 11, *a*). As soon as the apparatus enters the stomach and begins to float in the gastric contents, the lid drops down, opens the vessel, and allows fluid to enter (see Fig. 11, *b*). When it is pulled out, the orifice is closed.

Recently Einhorn[2] has described another apparatus without a lid (Fig. 11, *c* and *d*). This instrument consists of an olive-shaped vessel $\frac{11}{16}$ inch long and $\frac{5}{16}$ inch wide. The opening is wide, and above it is a handle to which a silver thread is attached. The whole apparatus is made of silver. The instrument is swallowed; the length of the thread, which is 16 inches, will show whether the instrument is in the stomach or not. It is allowed to remain in the stomach for five minutes and then withdrawn. If the stomach is not empty, a sufficient quantity of stomach-contents for ordinary purposes of analysis will be obtained in this way.

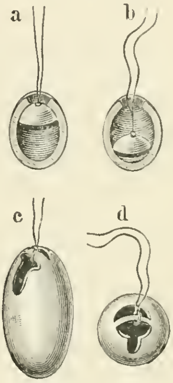

FIG. 11.—Einhorn's capsules.

It is self-evident that only enough stomach-contents can be obtained in this manner to perform a few of the hydrochloric acid reactions, and that the apparatus is altogether insufficient for more exact examinations.

All the methods described so far are intended for obtaining stomach-contents directly. Other investigators have followed a more indirect path in order to gain information in regard to the powers of the stomach. Günzburg[3] and Sahli[4] have devised such methods. Some substance like potassium iodid, which is rapidly absorbed, is placed into a fibrin capsule and swallowed. The time is determined when the first iodin reactions appear in the saliva, and conclusions are drawn herefrom in regard to the rapidity of fibrin digestion and the peptic power of the stomach.

Günzburg's method, in detail, is as follows: a tablet of potassium iodid (0.2 to 0.3) is inclosed in rubber tubes with very thin walls, and the ends of these tubes closed with hardened threads of fibrin. These are pressed into a gelatin capsule. The patient is first instructed to take a test-breakfast, and an hour afterward to swallow one of these capsules. The saliva is examined every quarter of an hour for iodin.

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[1] *Medical Record*, July, 1890.
[2] *Zeitschr. f. Krankenpflege*, November, 1894.
[3] *Deutsch. med. Wochenschr.*, 1889, No. 41.
[4] *Schweizer Correspondenzbl.*, 1891, No. 3.

Sahli's method is similar: 0.2 gm. of iodid of potash in pill form is placed into little rubber tubes consisting of Para rubber, and closed by a thread of fibrin. The bag is made water-tight, and placed in a gelatin capsule. The patient eats a test-meal at the same time that he swallows a capsule. Here, again, the saliva is examined for iodin every fifteen minutes.

No definite conclusions in regard to the hydrochloric acid of the stomach can be drawn from these experiments; at the same time these methods may be of value in certain cases, because they furnish some information in regard to the total digestion. They cannot, however, replace other methods of examination.

However clever these methods may be, therefore, they cannot replace aspiration of the stomach-contents for diagnostic purposes, nor can they furnish any information that we would not be able to obtain by the ordinary methods that are universally employed.

Methods for Stimulating the Secretion of Gastric Juice.—All authors seem to agree that the best time to examine the stomach-contents for practical purposes is at the height of digestion—that is, when gastric juice is being secreted by stimulation of the stomach-walls. Normally, this stimulation is due to what may be called digestive irritation. But this irritation is not the only way to cause secretion of gastric juice. In fact, in the original attempts to use the sound for diagnostic purposes all endeavors were directed toward methods for obtaining gastric juice free from food-particles.

Before describing the methods in use to-day for obtaining gastric juice I will say a few words in regard to the methods that were formerly employed for stimulating the secretion of this fluid. Leube, the founder of the method of diagnostic aspiration of stomach-contents, describes three methods for causing the secretion of gastric juice in an empty stomach:

1. Stimulation of Gastric Secretion by Mechanical Irritants.—Leube at first attempted to stimulate gastric secretion by feeding his patients with some mechanical irritant. He recommended barley-water. Leube, however, soon saw that this method did not give uniform results and was not applicable to every case, so he soon abandoned it.

2. Stimulation of Gastric Secretion by Chemical Irritants.—The stomach should either be empty or its contents be pumped out. The empty organ is then washed out with 400 c.c. (13½ fluidounces) of lukewarm water. All the fluid should be removed; it should be clear and neutral, and this, according to Leube, is always the case if the stomach contains no food-particles. Then some 50 c.c. (1.69 fluidounces) of a 3 per cent. solution of soda are poured into the stomach and allowed to remain there for twelve minutes. After this 500 c.c. (17 fluidounces) of lukewarm water are poured in. The fluid that runs out is poured back, so that the stomach-contents is thoroughly mixed with the water. A part of the fluid obtained in this manner is tested.

This method revealed that the stomach-contents of a normal person is neutralized by a soda solution remaining within the stomach for twelve minutes. In cases of disturbed gastric secretion the fluid is still more or less alkaline. A generalization of the results obtained showed that if the soda solutions, after remaining in the stomach for twelve minutes, were still alkaline, the gastric secretion of the patient was insufficient.

3. Stimulation of the Secretion of Gastric Juice by Thermic Irritants.—One hundred c.c. of iced water are poured into the stomach; the viscus must be empty and neutral. The cold water is allowed to remain in the stomach for ten minutes. The organ is then washed out with 300 c.c. (10 fluidounces) of water. In this procedure more than 300 c.c. (10 fluidounces) are always obtained. All this is tested for its reaction with litmus tincture or tropäolin. Thirty c.c. are acidified with the necessary amount of hydrochloric acid, and are tested for peptic digestion.

These are the methods devised by Leube. Neither one of them has been adopted. The last named is the one that Leube recommends particularly, but I[1] have compared it with the digestive method and have found that the dilution of the fluid is much too great to enable us to obtain any exact measurements for the amount of gastric secretion. It may, in addition, be impracticable to use iced water in certain cases.

The following method—electric irritation—is more scientifically interesting than practically useful.

Electric Irritation of the Stomach.—The attempt has been made to stimulate the secretion of gastric juice by electric irritants. Ziemssen[2] was the first to demonstrate by animal experiments that the percutaneous application of the galvanic current can increase the secretion of gastric juice. If a current was passed, a large amount of fluid was poured out. Rossi[3] performed similar experiments on dogs with gastric fistula. A part of his experiments are not altogether valid, because he touched the inner wall of the stomach with his electrode, so that mechanical irritation is not altogether excluded. At the same time he obtained similar positive results if the current was applied percutaneously.

Dr. Hoffmann[4] performed experiments on human beings in my clinic and at my suggestion. He limited himself to the employment of the galvanic current. We do not use the faradic current because it always produces contractions of the abdominal walls and thereby mechanical irritation. Some preliminary experiments that he performed on dogs with stomach fistula showed that the galvanic current exercises a certain influence on the secretion of gastric juice. Before applying the current only a few drops of a mixed fluid of neutral reaction were poured out: as soon as the current was passed, true gastric juice was secreted; when the current was interrupted, secretion continued for three minutes and then gradually stopped. Our experiments on human beings were performed at first on subjects that were not sufferers from stomach-disease. The one electrode—the anode—was placed over the back; the other smaller cathode was placed over the stomach region. I cannot enter into the details of this method, but it showed that subjects who secreted only a few drops of gastric contents when no current was passed, always secreted a large amount of gastric juice during galvanization.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxvi.

[2] *Klin. Vorträge*, No. 12.

[3] *Lo Sperim.*, 1881, quoted from *Virchow-Hirsch's Jahresbericht*, 1881.

[4] *Berlin. klin. Wochenschr.*, 1889, No. 13.

There can be no doubt, to judge from these experiments, that percutaneous application of the galvanic current, provided it is allowed to act for a sufficient length of time and is sufficiently strong, can stimulate the secretion of the gastric juice.

Einhorn[1] has performed a number of experiments on the effect of electricity, particularly from a therapeutic point of view. We will refer to his therapeutic results later on. Einhorn electrized the stomach directly by an electrode that he ordered the patient to swallow; the same physiologic results were obtained—namely, faradization of the stomach caused increased secretion during the time the current was passing; in other words, given similar conditions, a greater amount of acidity (caused by hydrochloric acid) was found if faradization was employed.

Allen A. Jones[2] showed that the electric current frequently caused an increase of the hydrochloric acid secretion in man. Apparently it does not always do this. Ewald,[3] together with Sievers, performed other experiments that demonstrated positively that faradization of the abdominal walls can influence the secretion of the stomach, particularly if the current is sufficiently strong and the electrodes are large.

However interesting all these results may be, they hardly promise to be of much practical value. A good method for studying the secretion of gastric juice in pathologic cases will never be furnished in this way.

Stimulation of the Gastric Secretion by Digestive Irritants.—Digestive irritants are the natural stimulants for gastric secretion. What we aim at chiefly in studying a case is to determine whether the stomach can elaborate the food, or in what direction perversions of its normal function exist. All the methods mentioned above fail to give us an insight into the digestive powers of the stomach. It is true that they seem to answer the question whether or not the stomach is capable of secreting gastric juice when stimulated by certain irritants; this, however, does not give us any information in regard to its normal powers, nor does it teach us whether and to what extent it elaborates food, whether or not its motor power is sufficient, etc. The only way to determine all these points is to stimulate the stomach to do the work that it is naturally fitted for by administering a test-meal.

In order to interpret the disturbances of chemism, motility, resorption, and of sensibility of the stomach that may be observed in pathologic cases after a test-meal, it is necessary to determine first what represents normal conditions in all these respects. Abnormal conditions can be studied only if the normal ones are fully understood. It is natural that the same test-meal should be given in all cases in order to have more accurate points of comparison.

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[1] *Berlin. klin. Wochenschr.*, 1891, No. 23; *Zeitschr. f. klin. Med.*, vol. xxiii., p. 369; *New York Med. Record*, November 9, 1889.

[2] *New York Med. Record*, 1891.

[3] *Loc. cit.*, p. 98.

The test-meal, moreover, should be adapted to the conditions of ordinary life, and should be composed of food that is similar to that eaten daily. Diagnostic examination of the stomach-contents is not intended to determine to what extent the stomach reacts to unaccustomed digestive irritants, but what it is capable of doing with an ordinary every-day diet.

I was the first to recommend a uniform test-meal. In the course of time many other test-meals have been recommended, the most important of which I will describe.

1. *The Test-meal Recommended by the Author.*—The patient is given a plate of meat-broth, a beefsteak weighing from 150 to 200 gm. (5 to 7 ounces), 50 gm. (1½ ounces) of mashed potatoes, and a roll. As we intend primarily to obtain some stomach-contents for examination, the stomach should be siphoned out within a few hours after the administration of this meal, and, on another occasion, as in Leube's experiments on the duration of digestion, seven hours later. The fact that the stomach is empty after seven hours does not demonstrate that gastric digestion is normal; this fallacy is exploded. Nothing is demonstrated by this excepting that there is good motor power.

There are pathologic conditions in which the stomach forces its contents into the intestine within the normal time or even before, but where chemism is [depressed or otherwise] abnormal.

It is well, therefore, to study each individual case and to find the most favorable time for aspirating the stomach-contents in each one. It is impossible to state in advance which will be the most favorable time; there are cases in which the stomach must be pumped out two or three hours after the meal; others in which it is better to wait for five or six hours. This constitutes a drawback to my method. As a rule, I empty the stomach four hours after the meal, provided that other indications are not present that determine me to select some other time. If the stomach is found empty after four hours, I know that the motor power of the organ is good; no conclusions, however, can be drawn in regard to its peptic powers. If the stomach is found empty after four hours, its contents should be withdrawn earlier the next day; if, on the other hand, a large quantity of coarse and only half-digested morsels of food are found after four hours, the examination on the next day should be made later. A single examination is never permissible.

2. *The Test-meal of Ewald and Boas.*—The patient, on an empty stomach in the morning, is given two rolls and a cup of tea, or from 300 to 400 c.c. (10 to 14 fluidounces) of water.

A test-meal of this kind is eminently suited for the purpose for which it is intended. All the substances that play a rôle in general nutrition are represented. One hour after the test-meal the stomach-contents is aspirated or evacuated by expression.

The administration of a test-breakfast and a midday test-meal are the two methods most employed. A great deal has been written in regard to the advantages of each. A polemic of this kind is worse than useless, as either method will lead to the goal. The one has advantages in one direction, the other in another. In certain cases one may be more appropriate than the other. Leube[1] seems to prefer the test-breakfast, although he recognized that the midday test-meal has the advantage of taking the every-day work of the stomach into consideration. The breakfast, on the other hand, seems to be more uniform.

A test-breakfast appears more appropriate to me because the examination can be made after so short a time,—that is, about an hour afterward,—whereas if the test-meal is employed, a much longer and a less constant time must elapse. In addition, certain patients may not be able to eat so large a meal, but are very well able to eat a small test-breakfast. I do not consider that the advantage of cleanliness that Ewald calls attention to is at all significant. It is hardly worth while discussing the relative advantages of washing out morsels of bread or particles of food that are left over from a test-meal. The question whether the one or the other is more appetizing is hardly one to be discussed, even though the latter may be malodorous as a result of certain digestive disorders; this can hardly be considered a drawback; on the contrary, it is an advantage, as it calls attention to the existence of certain pathologic processes.

On the other hand, the test-meal has certain advantages. The test-meal, for instance, demands more work from the stomach than the test-breakfast, so that the administration of a test-meal will give us a better insight into the real powers of the stomach. If a test-meal and a test-breakfast are given to the same patient for the sake of comparison, it will be found that the total acidity after the former is much higher; at the same time the value for free hydrochloric acid does not rise proportionately. For instance, a patient may still show free hydrochloric acid after the test-breakfast, but not after the test-meal. The only interpretation for this phenomenon seems to be that the stomach can secrete sufficient hydrochloric acid when a test-breakfast is administered to leave a residue of free hydrochloric acid, but not enough hydrochloric acid to digest the larger test-meal, and consequently no free or excessive hydrochloric acid is found.

It is possible that this observation, which I could verify in a number of cases, may explain certain differences in the results obtained by different investigators. A carcinoma case, for instance, in which the production of hydrochloric acid is not too low, may receive a test-breakfast from one physician, a test-meal from another, and the one find free hydrochloric acid and the other not. This shows that the results obtained by different investigators can be compared only if the experiments are carried out under identical conditions. We are certainly not able to compare the results obtained from the administration of a test-breakfast with those obtained from the administration of a midday test-meal.

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[1] *Diagnostik d. inneren Krankheiten.*

Jürgensen,[1] of Copenhagen, claims to have always found more total acidity after a test-meal than after a test-breakfast. He admits that the test-meal frequently causes a greater secretion of hydrochloric acid, but at the same time believes that the test-breakfast is the better meal. He does not believe that a test-meal in general gives better results than the test-breakfast, and the disadvantage of the test-meal is that it is always administered at the accustomed midday hour, so that the examination of the contents that is carried out several hours later must be performed with artificial light, and this he considers an unfavorable method of performing chemical examinations. In certain places, moreover, the midday meal is not taken before three o'clock in the afternoon, and this he believes is a serious drawback. The individual reader may attach whatever importance he wishes to these objections.

It is well to adapt the test-meal and the time of administering it to the ordinary habits of the patient. This will not always be possible, unless there were all kinds of test-meals, and this again would have certain disadvantages. Fleiner's[2] objection to tea is more or less justified, for it certainly constitutes an unaccustomed irritant for certain patients.

Fleiner, therefore, following the precedent of Kussmaul, administers a test-meal that is almost the same as that I have been in the habit of giving—namely, a plate of gruel, an order of tender beefsteak or roast-beef, and an adequate quantity of mashed potatoes.

I have already discussed another objection that Jürgensen formulates—namely, that it is more difficult under these conditions to select the time at which the stomach is most active.

In case it is desired merely to determine whether the stomach secretes an excess of hydrochloric acid, the test-breakfast is certainly sufficient; if, on the other hand, more than this question is to be answered,—if it is to be determined whether the stomach is capable of performing its ordinary functions,—the test-meal is better. I have been in the habit of employing both methods almost every day, and have arrived at the conclusion that the test-meal gives us a better picture of the total powers of the stomach than the test-breakfast. The test-breakfast, it is true, is more convenient, for we need not consider what is the best time for emptying the stomach, but simply wash it out one hour after the administration of the breakfast. A test-breakfast can be procured anywhere, and can even be administered during office hours. These are two great advantages, particularly as the test-breakfast in some instances is all that is needed if the hydrochloric acid question alone is to be decided.

The test-meal, on the other hand, gives us much better information in regard to the total results of digestion. This is apparent, for instance, if the stomach-contents of a case of ulcer, a case of carcinoma, or a case of continuous secretion of gastric juice are compared macroscopically after the administration of both these meals.

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[1] *Berlin. klin. Wochenschr.*, 1889, No. 20.

[2] *Volkmann's Samml. klin. Vorträge*, new series, No. 103.

3. The Method of Jaworski and Gluzinski.[1]—A preliminary examination of the stomach determines that no food is left in the organ. The patient is then given the whites of two hard-boiled eggs, without the yolks, together with 3 fluidounces of distilled water of room-temperature (64° F.). This meal is administered early in the morning on an empty stomach. The patient is then instructed to sit still for some time—from one to one and a quarter hours. After this time the stomach-tube is introduced and 3 to 10 fluidounces of distilled water of room-temperature (64° F.) are poured into the stomach in order to dilute its contents. The gastric contents is then aspirated with Jaworski's stomach aspirator until no more fluid can be procured; then more water is allowed to flow into the stomach. This is aspirated, and the process of washing continued until no more shreds of albumin appear in the gastric fluid. The first quantity is measured and at once examined chemically for its reaction, the presence of free hydrochloric acid, its digestive power after the addition of hydrochloric acid, for mucus, peptone, and syntonin.

This method presents no advantages over those above named.

4. The Method of Klemperer.—The patient, on an empty stomach, is given a pint of milk and two rolls. The stomach is washed out after two hours.

5. The Method of G. Sée.—The patient is given 3 to 5 ounces of bread, a large glass of water, and from 2 to 3 ounces of finely chopped meat.

6. *The Method of Bourget.*[2]—Five drams of toast, 5 ounces of weak tea without sugar, but with 4 c.c. (65 minims) of mint liqueur. The stomach is washed out from one to one and one-half hours after this meal.

All that has been said in regard to the first-described methods applies to the last-named ones. All the different methods lead to the goal, and are probably all sufficient to determine the hydrochloric acid secretion. If other questions are to be solved by this method of digestive irritation, the test-meal seems to be the most suitable method.

Bouveret is right when he says that my midday test-meal is somewhat inconvenient to administer. It is true that it is quite abundant, but I do not consider that it is too abundant; a diseased stomach can very well digest 5 to 7 ounces of beefsteak. It is also true that it is necessary to wait quite a while before washing out the stomach, but I do not consider this a disadvantage. I admit, for pathologic cases, Bouveret's objection that a large quantity of solid food constitutes an obstacle to aspirating the stomach, because the bread and the meat occlude the openings of the sound.. If digestion is normal, this does not occur. I see an advantage in being able to determine pathologic conditions by macroscopic examination of the stomach-contents and do not consider it a serious objection to my method that it may be occasionally necessary to remove the sound, get rid of a solid morsel of food, and reintroduce the instrument. These objections certainly do not overrule the advantages of my method, which Bouveret concedes—namely, the facility with which the different stages of digestion can be studied and with which the motility and the degree of retention can be judged.

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[1] *Zeitschr. f. klin. Med.*, vol. xi.

[2] *La méd. moderne*, August 4, 1892.

Sée's test-meal occupies an intermediate position between mine and that of Ewald-Boas, as it differs from my own only in the smaller quantity of meat that it contains.

I repeat that all methods are useful and all have their advantages and disadvantages. If it is desired to obtain results that can be utilized for comparison, it is necessary to use only one method. It would be very desirable if all clinicians could unite upon one method, so that the results obtained by different investigators could at once be compared.

[While the test-breakfast of Ewald and Boas has its place, so has the generous meal recommended by Riegel. But for practical purposes the most satisfactory test-meal is probably that recommended by Ewald, consisting of about 6 ounces of finely chopped meat, stale bread and butter, and a glass of water, to be taken three hours before the withdrawal of the stomach-contents. It is sometimes advisable to vary the quantity in proportion to the general activity of the patient. Taking the results found from the general examination of this meal as a standard, other foods may be allowed at a subsequent meal, and thus we may still further test the strength of digestion in a given case. This is very much like the method of Germain Sée.

Troller[1] repeated the work of Talma with Liebig's meat extract, found it of little practical value, and reached the same conclusion regarding Schule's method of obtaining the stomach-contents after the patient has chewed lemon-peel. He found, however, that when more stimulating substance—for instance, mustard—was chewed, a copious and concentrated gastric secretion was obtained, giving higher values both for hydrochloric acid and ferments than was secreted in normal gastric juice; when, however, an emulsion of mustard was introduced directly into the stomach, the gastric secretion was markedly diminished. He concluded that in the first instance the increased secretion resulted reflexly from the mouth through the sense of taste. In confirmation of this he found that tasteless substances when masticated led to the secretion of little gastric juice, whereas those that stimulated the sense of taste led to abundant secretion. He also discovered that in cases of hypochlorhydria chewing of spices led to the secretion of gastric juice even when a test-breakfast failed to do so; but in cases of achylia gastrica, both direct and indirect stimuli were without effect on the gastric secretion. Like Pawlow, Troller emphasizes the importance of the effect of taste in mastication upon the stimulation of gastric secretion, and showed that when food was introduced into the stomach through the stomach-tube, the secretion and the acidity were less than when food was masticated and swallowed.—Ed.]

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[1] *Zeitschr. f. klin. Med.*, vol. xxxviii., p. 183.

EXAMINATION OF THE STOMACH-CONTENTS.

The stomach-contents obtained by siphoning is expected to give us a clear picture of the amount of work performed by the stomach. We wish to find out whether the stomach has elaborated the food introduced in a normal manner or, if not, in what directions abnormal conditions exist. In order to answer the last question a chemical examination for hydrochloric acid alone is not sufficient, although many clinicians seem to believe this. Even the most exact quantitative determination of the free and the combined hydrochloric acid is insufficient for this purpose.

If we desire to obtain a correct idea of the amount of work performed by the stomach, it is necessary to analyze the stomach-contents in detail.

MACROSCOPIC EXAMINATION OF THE STOMACH-CONTENTS.

The first step in the examination of a test-breakfast or a test-meal should be macroscopic inspection. This may give us important information in regard to the quantity, the color, the consistency, the constitution of the morsels of food, the presence of abnormal admixtures, as mucus, blood, bile, etc. The quantity of stomach-contents aspirated naturally depends on the character of the test-meal, but also on the digestive powers of the stomach. Given the same disease, a test-meal will furnish a larger amount than a test-breakfast; the nature of the disease will also determine the quantity. In order to obtain a correct idea of the amount of material remaining in the stomach it is necessary to remove all the contents by aspiration.

In practice, simple siphoning or expression is sufficient. If necessary, the stomach can be washed out with water in order to determine whether anything has remained behind.

If it is desired to express in figures the total amount of fluid obtained from the stomach, the method of Mathieu and Remond[1] may be employed. These authors determine the amount of acid in the undiluted stomach-contents and then in the residue that they wash out with a certain quantity of water, and from these two figures calculate the quantity of material originally present in the stomach.

My former assistant Strauss[2] has described a method that is still more simple.

The specific gravity of the undiluted stomach-contents and of the stomach-contents after dilution with a known quantity of water is determined. If—

X = the amount of stomach-contents;
S = the specific gravity of the undiluted contents;
S_1 = the specific gravity of the diluted contents;
V = the amount of fluid aspirated;
a = the amount of water introduced;

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[1] *Soc. de Biol.*, 1890.

[2] *Therapeut. Monatsh.*, March, 1895.

the following formula can be calculated:

$$X = \frac{V.S + (a - V)\,S_1 - a}{S - S_1}.$$

This formula can be simplified if X is allowed to designate the quantity of fluid still remaining in the stomach and not the total quantity (Reichmann[1]). Under these conditions the formula is as follows:

$$X = \frac{a\,(S_1 - 1)}{S - S_1}.$$

Here the other letters have the same significance as above.

However desirable it may be to have methods of this kind in the solution of purely scientific questions, we certainly can get along without them very well in practice. All that the practitioner desires to know is the amount of fluid that he can remove by siphonage. In certain doubtful cases he may possibly gain additional information by washing out with water.

The quantity of residue obtained by siphonage varies greatly in pathologic conditions; in some cases it is normal; in others diminished; in still others increased. It is impossible to give any figures that would form the basis of a plus or minus. The practised eye will recognize at once whether there is too much or too little residue. We are never justified in drawing any conclusions in regard to the peptic powers of the stomach from the quantity of residue.

The quantity of residue merely determines the motor power of the stomach. We will discuss this more carefully when describing the methods for determining the motor power of the stomach. Motor and secretory powers of the stomach, however, do not run parallel; the secretion may be increased and the residue also be increased, or the secretion may be decreased and the residue be normal or even subnormal. In hypersecretion, for instance, we frequently see abnormally large quantities of stomach-contents, and inversely, in cases of subacidity, we see that the ingesta are moved into the intestine before the normal time limit. In fact, it seems sometimes as though the diminution in the secretion of gastric juice were, so to say, compensated by increased motor power. As an independent condition hypermotility is rarely seen.

We perceive, therefore, that the amount of residue does not permit any conclusions in regard to the secretory powers of the stomach. Inspection of the contents is more apt to give us this information.

Siphonage of the stomach gives us information chiefly in regard to the chemical conditions existing, and to a lesser degree in regard to the motor powers of the organ. All that would be needed to determine the motor powers of the stomach would be to wash out the stomach at a time when it should normally be empty. In the procedure we are describing, however, we desire to obtain stomach-contents at the height of digestion; for this reason siphonage must be performed at a time at which we are certain to find some residue in the stomach. At the same time this residue should be chemically changed. In the case of the test-breakfast, the most appropriate time is one-half hour after its ingestion; in the case of the test-meal the time varies in pathologic cases, as I have stated above.

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[1] See abstract in *Deutsch. med. Wochenschr.*, supplement, No. 12, 1895, p. 79.

If in pathologic cases siphonage reveals the presence of an abnormally large residue, we will only be justified in concluding that the stomach was unable to move the ingesta into the intestine in a normal manner. Subsequent examination will have to give us information in regard to its secretory power.

There are certain cases in which more stomach-contents is aspirated than was introduced. This is a very remarkable fact that every one has probably noticed who has examined many cases of stomach-disease, particularly cases of ectasy. This phenomenon seems incomprehensible, and it cannot apparently be reconciled with the view entertained until quite recently that the stomach normally possesses the power of absorbing large quantities of water; for even if the stomach did not absorb water and did not move the food onward, we should hardly expect to find the quantity of fluid larger a few hours after the introduction of a certain quantity of material, provided, of course, that no residue of some previous meal was present in the stomach. Von Mering[1] has shown, however, that the stomach does not absorb water, but, on the contrary, may excrete a certain quantity of water when substances like alcohol, sugar, dextrin, peptone, etc., are introduced, and that this secretion of water is, as a rule, proportionate to the amount of these substances absorbed. This will readily explain the peculiar phenomenon we have mentioned, particularly in cases of ectasy of high degree.

The macroscopic appearance of the stomach-contents is also important. I called attention to this fact many years ago. In order to accentuate the differences I allow the stomach-contents to flow into large graduated vessels; in this way the quantity can be determined and at the same time the color, the formation of different layers, the physical consistence of the particles contained in the stomach-contents, the presence of foreign admixtures, etc., can be studied.

As I have emphasized in another place, one of the chief advantages of my test-meal is that a more exact differentiation can be drawn between the digestion of carbohydrate and proteid material. In the test-breakfast, certain differences are observed; in some instances large undigested pieces of bread are brought up; in others remnants of bread that are only slightly digested or are almost completely digested; in other cases still there is a very fine, uniform, almost fluid, mushy contents. In the case of my test-meal the differences are more pronounced: in some cases a very fine, uniform, mushy liquid mass is seen that contains no coarse elements at all; in others again a mass of food containing many coarse pieces of meat that look as if they had just been swallowed; in addition there is frequently an abundant admixture of mucus. In some cases there is so much mucus that the food looks like a tough mass and passes through the sound with difficulty, and is very difficult to filter. In other cases there is a large quantity of fluid contents that forms three layers when kept in a glass vessel; at the bottom is seen a layer consisting of fine remnants of amylaceous material; above this a large layer of cloudy fluid, and on the top a foamy layer of different height. If the latter is present, it may be considered evidence of gaseous fermentation. This consistency of the stomach-contents is found chiefly in those cases in which there is stagnation or in which there is motor insufficiency. Gaseous fermentation is not dependent on the presence of lactic acid fermentation, which probably never occurs if considerable quantities of free hydrochloric acid are present; gaseous fermentation, on the other hand, is more frequently seen and is more intense in cases in which there is an abundant quantity of free hydrochloric acid.

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[1] *Therapeut. Monatsh.*, May, 1893.

Investigations by Kuhn[1] and Strauss[2] have shown that the hydrochloric acid of the stomach is unable to check fermentation by yeast; we can understand, therefore, that this form of gaseous fermentation may be present even if there are much hyperacidity and much hypersecretion, provided the conditions for stagnation of the stomach-contents are present. The admixture of blood, mucus, bile, and pus can frequently be seen on macroscopic examination.

We see, therefore, that macroscopic examination of the stomach-contents yields much important information; it tells us of the motor powers of the stomach, and to a certain extent of the secretory activity of the organ. If the mass removed consists of a fine uniform paste, we know that there is good peptic power or hyperacidity; if coarse morsels of meat appear, we know that there is subacidity; if there is an abundant quantity of amylaceous residue and at the same time evidence of good digestion of meat, we can diagnose hypersecretion.

Only chemical examination can give positive information in regard to the secretory powers of the organ. This alone, however, will not be sufficient, and the only way in which to gain full insight into the nature of the disease is to examine the stomach-contents in the several directions outlined.

If the food-remnants obtained from the stomach in different diseases are compared, the great significance of macroscopic inspection will be understood. In many instances this method alone will give us diagnostic points that we would otherwise obtain only by complicated chemical examinations. There are cases, for instance, in which the stomach-contents does not give any of the reactions for free hydrochloric acid. This shows that there is a deficit in the stomach. Sometimes, however, when free hydrochloric acid is absent we find only a relatively small amount of finely distributed food residue; at other times we may see larger quantities of coarse food-particles. If we limit ourselves to examining the filtrate in both these cases for free hydrochloric acid, we will probably consider that the two are alike, and, as a matter of fact, they are alike in regard to their free hydrochloric acid, for in neither do we see a formation of free hydrochloric acid. If, however, we consider the quantity and the appearance of the stomach-contents in both, we shall see that in the first case the peptic power is better than in the second.

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[1] *Zeitschr. f. klin. Med.*, vol. xxi., and *Deutsch. med. Wochenschr.*, 1892, No. 49.

[2] *Zeitschr. f. klin. Med.*, vol. xxvi., xxvii.

The first case is functionally nearly normal, for all the albumin has been digested; at the same time there was no residue of free hydrochloric acid. In the second case it is different; here the production of acid was subnormal, as shown by the disturbed digestion of meat. If this case is more carefully examined, it will be found that the deficit of hydrochloric acid is large, whereas in the first case it is small. In this way macroscopic examination frequently gives us a clear picture of disturbances of function that is much simpler and just as positive as the one obtained by complicated chemical examinations that are difficult to carry out in actual practice.

Frequently it is necessary to siphon the stomach in the morning when the organ is still empty, and it is well to wash out the stomach thoroughly on the preceding evening and to interdict all food during the night. This is necessary particularly in those cases in which hypersecretion is suspected. It is often possible to obtain considerable quantities of a cloudy, sometimes yellowish-green, bile-stained fluid that possesses all the properties of gastric juice. In other instances again nothing will be found in the empty stomach but mucus, or in cases of continuous regurgitation of bile, nothing but bile.

The odor of the stomach-contents is also important. Normal stomach-contents is odorless. In pathologic cases the stomach-contents may smell very acid or be offensive and putrid. If there is much butyric-acid fermentation, the characteristic odor of this acid will be noticed. If there is intestinal stenosis or some abnormal communication between the intestine and the stomach, the stomach-contents may have a fecal odor.

Ulcerative processes in other organs, as the esophagus, the mouth, etc., may cause decomposition of the stomach-contents and the production of a particular odor. It is never difficult, however, to differentiate between this secondary fermentation and the primary variety that occurs in the stomach alone.

CHEMICAL EXAMINATION OF THE STOMACH-CONTENTS.[1]—PURPOSE OF THE CHEMICAL EXAMINATION.

Short Historic Review of the Development of the Hydrochloric Acid Question.

Chemical examination of the stomach-contents should follow macroscopic inspection. Its purpose is to determine to what extent the chemical properties that the gastric secretion and the products of digestion should normally possess are present. By this examination we are frequently enabled to determine whether or not the stomach possesses digestive powers, and what abnormalities of this power exist.

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[1] Dr. Strauss, my former assistant, has aided me greatly in preparing this section, particularly in collecting the chemical literature on the subject. I wish to thank him in this place for his work.

The most important criterion from a practical point of view as to gastric secretion is the determination of an excess of hydrochloric acid at the height of digestion. The first question, therefore, that must be solved by chemical examination is whether or not there is present an excess of hydrochloric acid; this excess is called free hydrochloric acid. If free hydrochloric acid is present, it may be interesting to determine whether the secretion is normal or increased. In the latter instance we speak of *hyperaciditas hydrochlorica.*

If free hydrochloric acid cannot be demonstrated, the digestive power of the stomach is insufficient. We know this from experience. In the majority of cases this fact, coupled with the physical properties of the stomach-contents, is sufficient to determine, for practical purposes, the deficiency of gastric powers. In other cases of subacidity it may be important to determine the exact degree of the deficiency. This determination may be made in two ways: we can either determine the quantity of hydrochloric acid in combination or we can determine how much more hydrochloric acid should have been secreted in order to satisfy all the free affinities still present. The latter method is a very simple one and suffices for practical purposes.

We see, therefore, that the most important part in the chemical examination of the stomach-contents is the determination of free hydrochloric acid. We attach particular importance to the designation *free* hydrochloric acid in contradistinction to the words total hydrochloric acid and total acid. Many of the differences of opinion that exist in regard to this hydrochloric acid question can be traced to a confusion of these terms. The term hydrochloric acid alone should not be used at all. Unfortunately, many authors employ an inexact nomenclature to this day, although every one recognizes that not only from a scientific, but also from a practical, point of view the terms *free* hydrochloric acid and *total* hydrochloric acid should be strictly differentiated. In reviewing the history of the hydrochloric acid question it will be seen that much work was needed before the strict differentiation of the components of the gastric acidity was accepted; it took a long time before the clinical significance of free hydrochloric acid was appreciated and understood.

In the beginning only a few color-reagents—as methyl-violet, hydrochlorate of rosanilin, tropäolin 00—were employed for determining the presence or absence of hydrochloric acid in the stomach. Von den Velden[1] was the first to perform examinations of this kind in cases of gastrectasy, and to formulate a difference between gastric juice with free hydrochloric acid and gastric juice without it. In those forms of ectasy that were caused by a cancer of the pylorus he noticed the absence of hydrochloric acid, whereas in benign ectasies he could always demonstrate its presence. Von den Velden believed that in those ectasies in which the above-named color-reactions were negative no hydrochloric acid was secreted at all.

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[1] Von den Velden, "Ueber Vorkommen und Mangel der freien Salzsäure bei Gastrektasie," *Deutsch. Arch. f. klin. Med.*, 1879, vol. xxiii.

Ewald,[1] in the beginning, claimed that the anilin dyes employed by von den Velden for the determination of free hydrochloric acid were not suitable ones, and he also combated the conclusions that von den Velden drew from the results he obtained with these reagents. Investigations carried on in my clinic led to a different view of the matter. These were begun immediately after the appearance of von den Velden's report, and were carried on systematically.[2] We did not limit ourselves to the examination of cases of gastrectasy, but extended our investigations to all diseases of the stomach. In addition to testing the stomach-contents with color-reagents we instituted artificial digestion experiments with the gastric juice—*i. e.*, the filtrate of the stomach-contents. We soon demonstrated that the color-reactions were positive in all those cases where the digestive powers of the stomach were good. Wherever the color-reactions appeared, the gastric juice was capable of digesting albumin in a short time, and vice versâ.

On the other hand, we did not feel justified in accepting the theoretic view formulated by von den Velden in those cases in which the color-reactions and the digestion experiments were negative. We soon discovered that the addition of a quantity of free hydrochloric acid sufficient to produce color-reactions did not yield positive color-reactions if it was added to gastric contents that did not originally give these reactions and could not digest a disc of albumin. We drew conclusion from this discovery that the hydrochloric acid added was either changed or entered into some combination that rendered it undiscoverable by the color-reagents.

For some time these different color-reagents were universally adopted, and new stains were added to them. Some time later Cahn and von Mering[3] published a paper that seemed to invalidate the significance of all the color-reagents that were being employed. These investigators succeeded in demonstrating the presence of free hydrochloric acid in gastric juice that did not give the ordinary color-reactions, particularly the one with methyl-violet, and that, according to von den Velden and myself, did not contain free hydrochloric acid. If they removed all organic acids from gastric juice of this kind, they found an acid residue and demonstrated that its acidity was due to the presence of hydrochloric acid. They proved this by the cinchonin method, by which neutral chlorids are not decomposed, but by which the chlorin of hydrochloric acid is discovered. As the color-reactions were negative even in those cases in which hydrochloric acid was present, they declared themselves against the employment of methyl-violet and similar color-reagents, and stated, in opposition to von den Velden and myself, that hydrochloric acid was regularly present in the gastric juice of cases of carcinoma of the stomach.

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[1] Ewald, *Zeitschr. f. klin. Med.*, vol. i.

[2] Riegel, *Berlin. klin. Wochenschr.*, 1885; *Deutsch. Arch. f. klin. Med.*, vol. xxxvi.; *Zeitschr. f. klin. Med.*, vol. xi.; "Ueber Diagnostik und Therapie der Magenkrankheiten," *Volkmann's Samml. klin. Vorträge*, No. 289; Edinger, *Berlin. klin. Wochenschr.*, 1880, No. 9; *Deutsch. Arch. f. klin. Med.*, vol. xxix.; Kredel, *Zeitschr. f. klin. Med.*, vol. vii.

[3] Cahn and von Mering, "Die Säuren des gesunden und kranken Magens," *Deutsch. Arch. f. klin. Med.*, vol. xxxix.

These investigations by Cahn and von Mering almost shattered all confidence in color-reagents, and many clinicians were about to discard all color-tests and consider them as devoid of all diagnostic value, when von Noorden and Honigmann[1] succeeded in explaining the apparent contradiction that existed between my previous experiences and the results of Cahn and von Mering.

Honigmann and von Noorden started from the fact, which I had demonstrated for the first time, that every stomach-contents that gave distinct color-reactions was able to digest albumin rapidly, and that a stomach-contents that did not give these reactions was unable to change the disc of albumin. Cahn and von Mering's investigations, moreover, did not explain another fact—namely, that a mixture of hydrochloric acid in proportion sufficient to give positive color-reactions, if added to stomach-contents that did not give these color-reactions before, was still unable to cause the appearance of these reactions. Von Noorden and Honigmann repeated Cahn and von Mering's experiments, and found that all stomach-contents that did not give the color-reactions still contained an acid radicle that could redden litmus-paper, but that could not digest albumin. If they added to stomach-contents of this kind, the composition of which was determined, a titrated quantity of hydrochloric acid, they did not find the amount of hydrochloric acid that they calculated to find, but always discovered a deficiency of hydrochloric acid. This demonstrated that the acid residue of Cahn and von Mering was, in fact, hydrochloric acid, but not *free* hydrochloric acid; if it had been free, it would have been added to the quantity that was contained in the artificial mixture, and this increase should have been determinable. Honigmann and von Noorden, therefore, assumed that in all those cases in which color-reactions were absent a quantity of hydrochloric acid was present that was insufficient to satisfy all the affinities of the albumin of the food, and that, as a result, peptonization remained incomplete. These investigators determined the amount of this deficit by adding titrated hydrochloric acid to stomach-contents until color-reactions appeared; as soon as the color change occurred, they knew free hydrochloric acid to be present; in other words, they were certain that all hydrochloric acid affinities were satisfied.

In this manner the value of color-reagents for determining the presence of free, excessive, digestive hydrochloric acid was reëstablished, and at the same time an explanation was furnished for the fact that gastric juice that gave color-reactions could digest albumin, whereas gastric juice that did not give these reactions could not do so. In this manner, too, the necessity was first discovered of differentiating strictly between free and combined hydrochloric acid. The explanation was also given why stomach-contents that gave color-reactions had digestive powers, and why stomach-contents that did not give these reactions was not capable of digesting albumin.

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[1] Honigmann and von Noorden, "Ueber das Verhalten der Salzsäure im carcinomatösen Magen," *Zeitschr. f. klin. Med.*, vol. xiii.

All this did not establish how much hydrochloric acid had been used for digestion. Normal gastric juice, however, at the height of digestion always contains free hydrochloric acid in excess, so that if free hydrochloric acid was absent, a deficiency in the secretory powers of the stomach was demonstrated. It was an easy matter to determine the amount of this deficit by adding titrated hydrochloric acid until free hydrochloric acid appeared; in other words, until the normal conditions of the gastric juice were established. The amount of the deficit could readily be calculated from the amount of hydrochloric acid added.

I have intentionally given so much space to the investigations of my former pupils, Honigmann and von Noorden, because they are important and significant for the understanding of other investigations to be presently described; also because they have changed our views in regard to the hydrochloric acid question. In these investigations the difference in principle between free and combined hydrochloric acid was emphasized for the first time. On the basis of this work, different investigators attempted to find methods for determining the size of these two factors—namely, of free and combined hydrochloric acid. Of all methods devised, those of Sjöqvist[1], Leo[2], and Martius and Lüttke[3] are most commonly in use. At the same time there exists a difference of opinion in regard to the value of the different methods of determination and in regard to the application of these determinations to clinical facts. Only recently do we find more uniformity of opinion in regard to the clinical value of hydrochloric acid determinations, and I believe that this opinion essentially coincides with the views that I enunciated a long time ago and have maintained ever since.

The point of view adopted to-day is practically the following: The first question that the physician will ask is, Whether the digestive power of the gastric juice is good or bad. This question is answered by examining for free hydrochloric acid. For if free, or still more if excessive, hydrochloric acid is present at a given time after the ingestion of a certain amount of food, we will know that all hydrochloric acid affinities are satisfied, that the stomach has complied with all the demands on its chemical powers, and that it has succeeded in producing more hydrochloric acid than was needed. It is this free hydrochloric acid that we determine by our color-reagents; a normal stomach, therefore, produces an excess of hydrochloric acid, and if we can demonstrate the existence of this overproduction by our color-reactions, then we know that sufficient hydrochloric acid has been produced. In general, therefore, the determination of free hydrochloric acid will give us a great deal of information in regard to the normal digestive powers of the stomach. In healthy subjects this excess fluctuates within narrow boundaries, provided the conditions are the same. In pathologic cases this is different. Here we may see—(1) that hydrochloric acid is completely absent—that is, that there is no excess; (2) that there is only a slight excess; and (3) that there is an abnormal excess.

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[1] Sjöqvist, "Eine neue Methode, freie Salzsäure im Mageninhalt quantitativ zu bestimmen," *Zeitschr. f. physiol. Chemie*, 1888, vol. xiii., p. 1.

[2] Leo, "Eine neue Methode zur Säurebestimmung im Mageninhalt." *Centralbl. f. d. med. Wissensch.*, 1889, No. 26.

[3] Martius and Lüttke, *Die Magensäure des Menschen*. Stuttgart, 1892. (This contains a complete bibliography of the literature on the chemical analysis of the contents of the stomach published up to that time.)

We need hardly mention that for practical purposes it is unnecessary to make quantitative determinations of the hydrochloric acid in combination in cases 2 and 3; the presence of even slight quantities of free hydrochloric acid shows that the food is completely saturated therewith. In group 1 this is different; here it may be important and interesting to determine the quantity of hydrochloric acid in combination.

A second method to determine the degree of secretory insufficiency would be to determine the amount of the deficit.

These, in general, are the methods we shall have to adopt. The examination of the stomach-contents should be both *qualitative* and *quantitative.* In the first place we must determine whether free hydrochloric acid is present or not; after this is done we can advance to quantitative examinations—in the one group of cases, of free hydrochloric acid; in the other, of hydrochloric acid in combination or of the amount of the deficit up to the point where free hydrochloric acid appears.

DETERMINATION OF HYDROCHLORIC ACID.

The Qualitative Determination of Free Hydrochloric Acid.—The presence of free hydrochloric acid[1] is determined by a number of color-reagents. Many of the stains that have been recommended for this purpose are of historic interest only—among them Uffelmann's[2] red-wine stain, mallow-, raspberry-, and madder-stain.

Among the coal-tar colors we may mention methyl-violet; von den Velden[3] employed this stain for the first time, and a great many reports have at different times been published, all testifying to the utility of this stain in determining the presence of free hydrochloric acid in the stomach-contents.

Free hydrochloric acid changes the color of this stain from violet to blue; this change of color is sometimes difficult to determine; the reaction is, therefore, somewhat dependent on the subjective color-sense of the observer, and consequently not absolutely reliable; only a pronounced blue demonstrates the presence of free hydrochloric acid. We can state, from our personal experience, that a stomach-contents that stains methyl-violet blue also possesses good digestive powers for albumin; if the blue color appears, we know that at least sufficient free

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[1] The stomach-contents in the majority of cases is acid, so that it is unnecessary for the practitioner to test its reaction with litmus-paper; only in cases where the absence of free hydrochloric acid is established may the reaction of the stomach-contents be determined afterward.

[2] Uffelmann, "Ueber die Methoden des Nachweises freier Säuren im Mageninhalt," *Zeitschr. f. klin. Med.*, vol. viii., p. 393.

[3] *Deutsch. Arch. f. klin. Med.*, vol. xxiii.

HCl is present to digest albumin. For all these reasons methyl-violet is a reagent that commends itself for general use.

Benzopurpurin (von Jaksch[1]), malachite green (Köster[2]), smaragd green, and brilliant green (von Jaksch[3]) have never become popular. The same applies to ultramarine (Kahler[4]), sulphid of zinc (Kraus[5]), and Mohr's[6] and Reoch's reagents.

Congo-red and tropäolin 00 are the most popular stains of all the coal-tar series.

Congo-red was introduced by Hösslin[7] and myself[8] for the chemical analysis of the stomach-contents; it is a red benzidin stain that turns blue on contact with acids. It is a very simple and distinct reagent for free acids. If the color change occurs, there must be free acid; hydrochloric acid does not cause the change if combined with organic bases or albumin, nor with acid phosphates in solutions as dilute as those found in the stomach. It is true that a great many free organic acids may turn Congo-red blue in addition to hydrochloric acid. Quantities of these acids, however, large enough to produce this blue coloration are never found in the stomach-contents, so that the appearance of blue always indicates the presence of free hydrochloric acid.

Tropäolin 00 is also a good and safe reagent for free acid. I have employed it in watery solution for fifteen years, and have found that Merck's preparation is a very good one. Ewald[9] in the beginning doubted the value of tropäolin for demonstrating free hydrochloric acid. The reason for this was that the preparation he employed was not so good as ours. In a later report[10] he himself recommends tropäolin as the best reagent for free acid. Tropäolin, at the same time, is no better than Congo-red—in fact, it is not quite so sensitive; it reacts only to free acid; organic compounds of hydrochloric acid do not affect it.

Of late, Töpfer[11] has recommended 0.5 per cent. alcoholic solutions of dimethyl amidoazobenzol; according to the investigations carried on in my laboratory,[12] this reagent is very sensitive to free acids, but also reacts to moderately concentrated solutions of lactic acid, and, in addition, does not show so marked a color change when it does react.

All the above-mentioned stains possess the property of reacting with all free acids, including the organic acids. They are not so sensitive, however, to free organic acids as they are to free mineral acids; or, in other words, the concentration of free organic acids must be much greater to bring about the reaction than that of free mineral acids. Thus, for instance, a 0.01 per cent. solution of hydrochloric acid will produce a distinct reaction with Congo-paper (Martius[1]), and three times as much lactic acid is needed to produce the same result. In my experience, I have never seen a case in which lactic acid or other organic acids were present in the stomach in quantities large enough to produce the reaction, so that any error that might be attributed to the presence of organic acids can be neglected. At the same time even a remote possibility of error may be eliminated by employing two reagents that react only to free mineral acids; these are Günzburg's[2] and Boas'[3] reagents.

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[1] Von Jaksch, *Klin. Diagnostik der inneren Krankheiten*, third edition, 1892.

[2] Köster, "Upsala Läkarefören," *Förhandling*, 1885, Nos. 5, 6.

[3] Von Jaksch, *loc. cit.* [4] Kahler, *Prager med. Wochenschr.*, 1887, No. 32.

[5] Kraus, *ibid.*, 1887, p. 439.

[6] Mohr, *Lehrbuch der chemisch-analytischen Titrirmethode*, Braunschweig, 1878.

[7] Von Hösslin, "Ein neues Reagens auf freie Salzsäure," *Münch. med. Wochenschr.*, 1886, No. 6.

[8] Riegel, "Ueber die Indicationen zur Anwendung der Salzsäure bei Magenkrankheiten," *Deutsch. med. Wochenschr.*, 1886, No. 35.

[9] *Zeitschr. f. klin. Med.*, vol. i.

[10] Ewald and Boas, "Beiträge zur Physiologie und Pathologie der Verdauung," *Virchow's Arch.*, vol. ci.

[11] Töpfer, "Eine Methode zur titrirmetrischen Bestimmung der hauptsächlichsten Factoren der Magenacidität," *Zeitschr. f. physiol. Chemie*, vol. xix., No. 1.

[12] Strauss, "Zur quantitativen Bestimmung der Salzsäure im menschlichen Magensaft," *Deutsch. Arch. f. klin. Med.*, vol. lv.

Günzburg's reagent consists of 2 gm. of phloroglucin, 1 gm. of vanillin, and 30 gm. of absolute alcohol. Boas' reagent consists of 5 gm. of resorcin, 3 gm. of cane-sugar, 100 gm. of distilled alcohol.

Günzburg's reagent is employed a great deal; Boas' reagent is not used so much.

Method of Employing the More Common Reagents for Free Hydrochloric Acid.—We will limit ourselves to describing the method of using the more common reagents for hydrochloric acid—namely, methyl-violet, Congo-red, tropäolin, Günzburg's, and Boas' methods.

In using methyl-violet two test-tubes of equal caliber are filled to one-third with a very dilute (about 0.2 pro mille) watery solution of methyl-violet. Then a few cubic centimeters of filtered stomach-contents are allowed to flow into one of the test-tubes. The color of the two is then compared. It is often well to add as much distilled water to the second test-tube as stomach-contents to the first, so that an equal amount of fluid is present in both tubes and the dilution of the stain is the same. If free hydrochloric acid is present, the violet tint of the solution will turn to blue.

The reaction may also be performed as follows: A little methyl-violet solution is poured into a porcelain dish, and the dish moved about so that a thin violet layer is formed on the bottom; if a drop of the stomach-contents filtrate is allowed to flow from the margin of the porcelain dish, a distinct blue zone will be formed at the point of contact of the two liquids, clearly distinguishable from its violet surroundings. If there is a pronounced blue tinge, we know that free hydrochloric acid is present.

Tropäolin 00 may be employed in the same manner, either in an alcoholic solution or better in a concentrated watery solution. Free hydrochloric acid turns the yellow color of these solutions into a deep red. I am in the habit of using the watery solution alone. Sometimes tropäolin is used in the shape of paper saturated with the stain in the same manner as Congo-red.

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[1] Martius, *loc. cit.*, p. 42.

[2] Günzburg, "Neue Methode zum Nachweise freier Salzsäure im Mageninhalt." *Centralbl. f. klin. Med.*, 1887, No. 40.

[3] Boas, "Ein neues Reagens zum Nachweis freier Salzsäure im Mageninhalt," *ibid.*, 1888, No. 45.

Congo-paper[1] is filter-paper saturated with a watery solution of Congo-red. It is pale pink in color. A strip of this paper is dipped into filtered or non-filtered stomach-contents; if free hydrochloric acid is present, the paper will turn blue, and the moist blue part will be distinctly differentiated from the red and dry part. As moistening the paper sometimes produces a slight change in the shade of red, it is well to control the experiment by moistening another strip of Congo-paper with distilled water and comparing the two. This is necessary, however, only when the reaction is far below normal. If appreciable quantities of free hydrochloric acid are present, the color change will be very marked.

Günzburg's reaction is performed as follows: One or two drops of the reagent are put into a small porcelain dish, the lid of a porcelain crucible, or a porcelain spoon, and mixed with the same quantity of stomach-contents. The two fluids are carefully heated over a small flame; it is not well to bring the flame too close to the porcelain dish. The heating should be interrupted from time to time and the dish kept moderately cool by blowing. These precautions are necessary, because too great heating of the fluid causes it to evaporate and burns the stain. As soon as the fluid evaporates, a red marginal zone will be seen if free hydrochloric acid is present; this will gradually spread until it involves the whole moistened surface. Reaction is positive only if the color is an intense red. Brownish or yellowish colors may be caused by drying or partial carbonification of albumin.

Günzburg's reagent should be prepared fresh from time to time and should be kept in a brown bottle, as it readily decomposes and forms a sediment.

Paper saturated with Günzburg's reagent has been recommended. It should be used as follows: The paper is moistened with a few drops of gastric contents and heated as above in a porcelain dish. This method of employing the reagent has no advantages.

Boas' reaction is also performed by allowing the two fluids to evaporate. The shade of red is slightly different—more like cinnabar.

Of all the reagents that we have described, the best ones are Congo-paper and Günzburg's reagent. These two are the most universally employed.

Günzburg's reagent, it is true, gives more uniform results than Congo-paper, as the former reacts only to free hydrochloric acid, whereas Congo-paper also reacts to other free acids. They are both equally sensitive to free hydrochloric acid.

On the other hand, Congo-paper is simpler than Günzburg's reagent. No apparatus is needed for warming and evaporating; any practitioner can carry a few strips[1] with him and perform his reactions at the patient's house, either with filtered or non-filtered gastric juice or with a drop of the fluid that he finds in the lower opening of the stomach-tube. In doubtful cases, if the reaction is very weak, it may be well to control the results with Günzburg's reagent. It will be very rare indeed—and personally I have never seen such a case—to find such large quantities of lactic acid that Congo-paper is colored blue. If this should ever occur, it is probable that such abnormally large quantities of lactic acid would immediately be revealed by the smell and the appearance of the stomach-contents and would naturally call for a special examination. [Lactic acid is not to be detected by its odor, nor does the appearance of the stomach-contents afford a reliable guide to its presence.—Ed.]

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[1] Unfortunately, the Congo-paper of commerce is not uniformly sensitive. It would be desirable to have it always made with solutions of the same concentration. Hübner informs me verbally that the best concentration for saturating paper is 1 : 1000.

If we see a pronounced reaction with Congo-paper or Günzburg's reagent, we know that the stomach has secreted sufficient hydrochloric acid;[2] with a little practice it will even be possible to determine, from the intensity of the reaction, whether the quantity of the free hydrochloric acid is normal or subnormal. Those who are not practised may perform exact determinations by making one of the quantitative examinations that we will describe below. [Even the most practised should resort to the quantitative methods with rare exceptions.—Ed.]

If the color-reactions are negative, we may say that the stomach is not capable of normal digestion. I base this statement on the results of the digestive experiments that I have performed.[3] This is the most important fact to be elicited, and the other questions, whether the stomach contains much, little, or no hydrochloric acid in combination, or whether much hydrochloric acid must be added in order to cause the appearance of free hydrochloric acid, are of subordinate importance [but still of importance]. For the diagnosis and for the treatment of these cases of subacidity the most important point to elicit is whether large quantities of foreign acids, particularly lactic and volatile acids, are present.

The Quantitative Determinations of Hydrochloric Acid.—The most important questions remain, whether or not free hydrochloric acid is present and whether or not the stomach-contents gives distinct reactions for free hydrochloric acid. If these questions are once decided, it may still be interesting to perform quantitative acid determinations. Such a determination is necessary in cases where the presence of free hydrochloric acid is demonstrated by the color-reagents.

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[1] By my request the chemical factory of Merck manufactures small booklets of Congo-paper similar to the litmus-paper books that have been in use for some time.

[2] We may say that, almost without exception, a sufficient quantity of pepsin is present whenever free hydrochloric acid is found. Up to date only a few isolated observations are on record in which there were excessive secretion of hydrochloric acid and a relative decrease in the secretion of pepsin, causing, of course, disturbances in the digestive powers. (See Sticker, *Münch. med. Wochenschr.*, 1886, Nos. 32, 33, and Gerhardt, *Berlin. klin. Wochenschr.*, 1886, No. 36.)

[3] I advise performing artificial digestion experiments in cases of this kind. If all investigators had done this as I have, there would have been less confusion in regard to the value of color-reagents.

If the color-reactions are negative, we know that the production of hydrochloric acid is, at all events, reduced. In a case of this kind it may be important to determine the presence of organic acids and to determine how large the deficiency of hydrochloric acid is. Other quantitative determinations are of little practical value. It is important, however, to determine the quantity of free hydrochloric acid present and to decide whether it is normal or increased.

Quantitative determinations should be made in three directions: (1) For the total acidity; (2) for the amount of free hydrochloric acid; (3) for the amount of hydrochloric acid in combination.

The quantity of organic acids, particularly of lactic or of acid salts, may also be determined by quantitative methods.

(a) **Determination of the Total Acidity.**—The total acidity is caused by the following factors: First, free hydrochloric acid; second, hydrochloric acid in combination; third, acid salts; fourth, organic acids.

The determination of the total acidity is made with $\frac{1}{10}$ normal sodium hydrate solution; phenolphthalein in 1 per cent. alcohol solution is used as an indicator; this body turns red as soon as the reaction of the fluid becomes slightly alkaline. The other indicators, as litmus tincture, rosolic acid, etc., do not indicate the transition from acid to alkaline reactions so distinctly, and consequently do not yield such positive results on titration.[1]

The determination is carried out as follows: 10 c.c. of filtered stomach-contents are measured in a pipet or a measuring flask. This quantity is poured into a beaker; three or four drops of phenolphthalein solution are added, causing a slightly grayish clouding. One-tenth normal sodium hydrate solution is then allowed to flow from a buret until the reddish color of the fluid no longer disappears, when the mixture is agitated. The beginning of the reddish color is seen very well if the beaker is placed on a white foundation—a porcelain plate. Ten c.c. of the filtrate are usually employed, but the test can be performed with less—for instance, 5 c.c.

Jaworski[2] and Ewald have proposed calling the number of cubic centimeters of $\frac{1}{10}$ normal sodium hydrate solution that are needed to neutralize 100 c.c. of stomach-contents the degree of acidity, instead of calculating the acid equivalent of the amount of soda solution used for neutralization. If, for instance, 5.8 cm. of $\frac{1}{10}$ normal sodium hydrate solution are needed to neutralize the stomach-contents, a corresponding hydrochloric acid equivalent is not calculated, but the acidity of the stomach-contents is called 58.

If any one desires to know the exact quantity of hydrochloric acid, it is an easy matter to calculate it according to the well-known laws of molecular equivalents: 1 c.c. of $\frac{1}{10}$ normal sodium hydrate solution corresponds to 3.65 mg. of hydrochloric acid. In the example quoted above there were, therefore, 59 times 3.65 mg. = 211.7 mg. of hydrochloric acid in 100 c.c. of gastric contents, or, calculated for 1 gm. or 1 cm., 2.1 pro mille of hydrochloric acid.

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[1] It is always well to use the same indicator, as otherwise errors of several cubic centimeters may occur.

[2] See *Zeitschr. f. klin. Med.*, vol. xi., p. 54; and *Deutsch. med. Wochenschr.*, 1887, Nos. 36–38.

Titration of the filtered stomach-contents until the change of color of the phenolphthalein indicator is brought about gives us the total acidity, or, in other words, the amount of acid factors present in the stomach-contents. We know from this determination how many acid valences are dissolved in the stomach-contents. In many cases it is interesting to know what the quantities of the different acid components of the stomach-contents are—for instance, free hydrochloric acid, hydrochloric acid in loose combination, acid salts, and organic acids.

Of these different components of the total acidity, the quantity of free hydrochloric acid is practically the most important. It is particularly interesting to determine free hydrochloric acid in those cases where there is a suspicion of *hyperaciditas hydrochlorica.*

However true it may be that hyperaciditas hydrochlorica exists only in those cases in which the quantity of free hydrochloric acid is abnormally large, this conception really bases on something else. Many investigators have spoken of hyperacidity in general where there was a distinct hydrochloric acid reaction and the total acidity was very high. In examining the hyperacid forms the color-reactions were made and the total acidity determined. In those cases where the reaction for free hydrochloric acid and for free acid is pronounced, we know that appreciable quantities of organic acids play no rôle, and we are, therefore, justified in attributing the total acidity in these cases to the abnormal amount of hydrochloric acid present. Even to-day the majority of investigators are content with determining the increased total acidity and pronounced positive color-reactions ; if these two tests are positive, they speak of hyperacidity—in fact, nearly all the figures for hyperacidity published in the reports of recent authors refer to the total acidity and not to the quantity of free hydrochloric acid. At the same time it should be remembered that, strictly speaking, hyperaciditas hydrochlorica exists only in those cases in which not only the total acidity, but the free hydrochloric acid, is known to be increased.

(b) Quantitative Determination of Free Hydrochloric Acid.—Mintz[1] calculated the amount of free hydrochloric acid present in a definite quantity of filtered stomach-contents by allowing $\frac{1}{10}$ normal sodium hydrate solution to flow into the filtrate until the Günzburg reaction for free hydrochloric acid could no longer be obtained. Mintz based his method on the supposition that the normal sodium hydrate solution would first combine with the free hydrochloric acid and then with the hydrochloric acid in organic combinations. If, for instance, Günzburg's reaction appears in 10 c.c. of stomach-contents after the addition of 1.2 c.c. of normal sodium hydrate solution, but if it no longer appears on addition of 1.3 c.c., the quantity of free hydrochloric acid present, according to Mintz, corresponds to 12 + 1 c.c. of normal sodium hydrate solution; in other words, the acidity of free hydrochloric acid is 13 c.c., or 0.047 per cent. of free hydrochloric acid.

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[1] Mintz, "Eine einfache Methode zur quantitativen Bestimmung der freien Salzsäure im Mageninhalt," *Wien. klin. Wochenschr.*, 1889, No. 20.

It is technically important not to remove fluid with a glass rod, but with a platinum loop, in performing Günzburg's reaction, because if the latter method is employed, there is less loss of the substance and the results of the titration are more exact. Ewald[1] indorses this recommendation and also advises employing Günzburg's reaction.

Fleiner[2] also employs Mintz's method. He advises the following procedure: 25 to 30 drops of Günzburg's reagent are added to 10 c.c. of stomach-contents; a drop of this mixture is placed into a porcelain spoon and carefully heated. If a red mirror appears, this demonstrates the presence of free hydrochloric acid. In order to determine the free hydrochloric acid quantitatively, $\frac{1}{10}$ normal sodium hydrate solution is allowed to drop into the mixture until heating of a small quantity no longer gives a red mirror. The amount of free hydrochloric acid is indicated by the number of cubic centimeters of the $\frac{1}{10}$ normal sodium hydrate solution that were necessary to bind the free hydrochloric acid. Following this titration the total acidity can be determined by titration with phenolphthalein as an indicator.

Mörner[3] determines the amount of free hydrochloric acid present in the filtrate by the same principle, but uses Congo-red as an indicator. We have employed this method for years in our clinic.

Another method for determining the amount of free hydrochloric acid is to dilute the filtrate with distilled water until Günzburg's reaction no longer appears. The calculation is based on the limit of Günzburg's reaction, which we know empirically to be 0.05 hydrochloric acid in 100 c.c. of stomach-contents = 0.0365 of hydrochloric acid in 100 c.c. of stomach-contents (Mintz).

Boas[4] does away with the removal of drops by adding watery Congo-solution to the fluid that is to be titrated. Martius[5] employs a solution of tropäolin (1 to 10 in dilute alcohol) for the same purpose, and Töpfer[6] uses a 0.5 per cent. alcohol solution of dimethylamidoazobenzol. Boas's modification is a simple one, and the employment of tropäolin and of dimethylamidoazobenzol further simplifies the titration. One drawback to titration with Congo-solution is, according to Boas, that the coloring-matter of Congo forms a fine blue-black sediment that remains suspended in the fluid as long as free hydrochloric acid is present; this obscures the distinctness of the reaction. The method with dimethylamidoazobenzol has the disadvantages that we have already discussed, and the tropäolin solution is not so sensitive as Congo-red.

In my experience the method of moistening Congo-paper with stomach-contents is the best. It is better than Mintz's method, because it is more rapid and more simple. As a rule, the free hydrochloric acid and the total acidity are determined as follows in the same specimen of stomach-contents :

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[1] Ewald, *Klinik der Verdauungskrankheiten*, third edition, vol. ii., p. 47.

[2] Fleiner, "Erfahrungen über die Therapie der Magenkrankheiten," *Samml. klin. Vorträge*, new series, 1894, No. 103.

[3] "Simple Method of Determining the Power of the Stomach to Secrete Hydrochloric Acid," from Maly's *Jahresb. f. Thierchemie*, vol. xix., p. 253.

[4] Boas, *Centralbl. f. klin. Med.*, 1891, No. 2.

[5] Martius and Lüttke, *loc. cit.*, p. 67.

[6] Töpfer, *loc. cit.*

First, we determine the presence of free hydrochloric acid by quantitative tests ; then 10 c.c. of the filtrate are used for determining the total acidity. At first we do not add phenolphthalein ; $\frac{1}{10}$ normal sodium hydrate solution is allowed to drop from the buret while we remove a drop of the mixture from time to time with a platinum loop until Congo-paper is stained bluish violet. As soon as the change of color in the Congo-paper becomes indistinct, we control this color change by dropping distilled water on the same piece of Congo-paper. The number of cubic centimeters of $\frac{1}{10}$ normal sodium hydrate solution that had to be added until this reaction occurred indicates the amount of free hydrochloric acid present in the stomach-contents. This figure is commuted for 100 c.c. of stomach-contents, so that we see that the gastric juice contains 30 c.c. free hydrochloric acid in case we use 3 c.c. of $\frac{1}{10}$ normal sodium hydrate solution. Now we add phenolphthalein solution, and continue to titrate in the same manner as described above in the determination of the total acidity, until the color of the solution turns red. The total acidity is indicated by the total quantity of cubic centimeters of $\frac{1}{10}$ normal sodium hydrate solution used from the beginning of the titration. If, for instance, 6.8 c.c. have been used, the total acidity of the stomach-contents is 68 ; 30 of this acidity are due to free hydrochloric acid.

The principle of this method is, therefore, the same as that of Mintz, with this difference, however, that in the former method Congo-paper is used for the quantitative determination of free hydrochloric acid, whereas in the latter Günzburg's reagent is used. This is followed in both cases by the quantitative determination of the total acidity with phenolphthalein.

(c) Quantitative Determination of the Total Hydrochloric Acid and of Hydrochloric Acid in Combination.—After having determined the total acidity and the amount of free hydrochloric acid, the difference between the two figures obtained indicates the amount of acid residue, which is composed of hydrochloric acid in combination, acid phosphates, and possibly the organic acids.

If a large amount of free hydrochloric acid is present, the quantity of organic acids that are usually formed by fermentative processes is so small that from a practical point of view exact quantitative determinations of these organic acids are essentially fictitious.

It might be possible to determine them as follows : The lactic acid might be extracted by ether, and the residue titrated after the volatile acids had been evaporated. On the other hand, the lactic acid might be determined and the volatile acids estimated from the distillate or indirectly by calculation.

The determination of the acid phosphates is not important from a practical and diagnostic point of view ; at the same time we should not underestimate their significance in the total acidity. We will describe below a method evolved by Leo for determining them.

Very much has been written in regard to the clinical significance of combined hydrochloric acid in the presence or absence of free hydrochloric acid. There has been a considerable polemic in regard to the best and simplest method for determining hydrochloric acid in combination. In order to discuss this question we must first describe the methods that are employed for determining hydrochloric acid quantitatively, for a great many of these methods are based on determinations of all the hydrochloric acid of the stomach-contents that is not combined with alkalis. If free hydrochloric acid is absent, the figure found indicates the hydrochloric acid that is combined with organic bases, chiefly proteids, whereas if free hydrochloric acid is present, the figure obtained as above must be subtracted from the figure for free hydrochloric acid. We cannot emphasize too strongly that in the presence of free hydrochloric acid the most interesting and important question is the quantitative determination of the amount of free hydrochloric acid ; accordingly, the whole question in regard to the best method for the determination under discussion depends on the presence or absence of free hydrochloric acid. Only in the absence of free hydrochloric acid does a determination of the total acidity answer this question. If free hydrochloric acid is present, the titration of free hydrochloric acid is hardly less important than the titration of the total acidity.

There are many methods for the determination of free, plus combined, hydrochloric acid, which Martius groups under the name of physiologically active hydrochloric acid (as free hydrochloric acid is still capable of aiding digestion, whereas the hydrochloric acid in combination has already performed this function).

New methods are invented almost daily, so that there are nearly a dozen that are of value. When many of these will lead to the same goal, we should always prefer the safest and shortest one.

In the following we will discuss only those methods of examination that always lead to positive and reliable results, and that are not too difficult. Those methods particularly will be considered that are practical —that is, that answer questions that are important chiefly in diagnosis and treatment.

The oldest method employed is that of Bidder and Schmidt.[1] Here the total quantity of chlorin present in a stated quantity of stomach-contents is first determined, then the amount of inorganic bases that are combined with chlorin to neutral salts. If the amount of chlorin that is combined with these bases to salts is deducted from the total quantity of chlorin found, the figure obtained will indicate the amount of chlorin present as hydrochloric acid, either free or in combination. It is impossible to separate free from combined hydrochloric acid by this method. The same objection obtains in the method of Hehner and

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[1] Bidder and Schmidt, *Die Verdauungssäfte und der Stoffwechsel*, Mitau and Leipsic, 1852.

Seemann,[1] which is identical with the method of Braun, which Leube recommends. This method is relatively simple and easy, and has the advantage of enabling us to determine the amount of hydrochloric acid indirectly by incinerating the stomach-contents and in this way determining the organic acids present in the gastric juice. If the figure found for organic acids is deducted from the total acidity, the figure obtained will indicate the quantity of free plus combined hydrochloric acid.

According to Hehner and Seemann, this test is executed as follows: To a measured quantity (10 c.c.[2]) of filtered stomach-contents $\frac{1}{10}$ normal sodium hydrate solution is added until the mixture is neutral; then the liquid is evaporated to dryness, and the residue incinerated over a free flame. The evaporation is performed either on the water-bath or on a copper or asbestos plate. The ash consists of neutral salts and alkali carbonates. The latter are determined by extracting the ash with hot water as long as the extracts show an alkaline reaction. This extract is then titrated with $\frac{1}{10}$ normal acid ($\frac{1}{10}$ normal sulphuric acid is the best), and the figure obtained indicates the amount of organic acid present in the solution. The difference corresponds to the free or combined hydrochloric acid that was present.

Kossler[3] claims that by this method the values obtained for hydrochloric acid are too high, as the acid phosphates lead to wrong results.

Braun's modification of this method is only slightly different. The total acidity of a certain quantity of filtered stomach-contents is again determined by titration with $\frac{1}{10}$ normal sodium hydrate solution, then $\frac{1}{10}$ normal sodium hydrate solution in excess is added to a second quantity of stomach-contents that is just as large (5 or 10 c.c.). This mixture is again evaporated to dryness, and the residue incinerated as above. The ash is now dissolved in as many cubic centimeters as cubic centimeters of normal sodium hydrate solution were employed to alkalinize the specimen. The solution is then heated in order to drive off free carbon dioxid, and titrated with $\frac{1}{10}$ normal salt solution, using three drops of phenolphthalein solution as an indicator. If, in this test, x cm. of a $\frac{1}{10}$ normal sodium hydrate solution were employed, this corresponds to the amount of hydrochloric acid (free + combined) of $x \times 0.00365$ pro 5, or 10 c.c. of the solution).

The methods of Cahn and von Mering,[4] the method of Hoffmann,[5] and the method of Jolles[6] are no longer universally employed, chiefly because they are too complicated.

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[1] Seemann, "Ueber das Vorhandensein freier Salzsäure im Magen," *Zeitschr. f. klin. Med.*, vol. v., p. 272.

[2] Leube, *Specielle Diagnose der inneren Krankheiten*, third edition.

[3] Kossler, "Beiträge zur Methodik der quantitativen Salzsäurebestimmung im Magen," *Zeitschr. f. physiol. Chemie*, vol. xvii., p. 91.

[4] Cahn and von Mering, "Die Säuren des gesunden und kranken Magens," *Deutsch. Arch. f. klin. Med.*, vol. xxxix., p. 239.

[5] Hoffmann, "Erkennung und Bestimmung der Salzsäure im Magensaft," *Centralbl. f. klin. Med.*, 1889, No. 46.

[6] Jolles, "Eine neue quantitative Methode zur Bestimmung der freien Salzsäure des Magensaftes," *Wien. med. Presse*, 1890, No. 51, and Jolles, "Einfacher Apparat zur quantitativen Bestimmung der freien Salzsäure im Magensaft," *Wien. med. Wochenschr.*, 1891, No. 22.

The method of Cahn and von Mering consists in successively removing the volatile acids and lactic acid, and in making the hydrochlorate of cinchonin. This salt is isolated, and the chlorin it contains determined. The method has chiefly a historic interest, because it was extensively employed at the time when the hydrochloric acid question was being acutely discussed. In the course of time, however, it has been replaced by more practical and simpler methods.

The method of F. A. Hoffmann for determining the hydrochloric acid is based on the property of hydrochloric acid to convert solutions of cane-sugar into invert-sugar. After this inversion the rotary powers of the solution are changed. Hoffmann's method is scientifically very interesting, but much too complicated for practical purposes.

Of the two methods of Jolles, one is based on the property of solutions of eosin to show two absorption-bands in the blue-green part of the spectrum if the solution is alkaline or neutral, and on the disappearance of these bands on addition of a few milligrams of either free or combined hydrochloric acid. This method, therefore, indicates only the total hydrochloric acid.

The other method of Jolles, also a colorimetric one, is executed as follows: The stomach-contents containing hydrochloric acid is allowed to drop into a solution of brilliant green, and the different shades obtained compared with a number of colored glass plates whose shades correspond to hydrochloric acid solutions the titration value of which has been empirically determined. This method is a simple one, but has the disadvantage, like any other colorimetric method, of being dependent on subjective impressions.

The method of Sjöqvist[1] merits more careful discussion. It consists in transforming free and combined hydrochloric acid into barium chlorid by incineration with barium carbonate, and determining the amount of barium chlorid by a number of different methods.

Sjöqvist's method is executed as follows: A small quantity of barium carbonate is added to 10 c.c. of filtered stomach-contents, the mixture evaporated and heated to faint glowing; hereby barium chlorid, barium carbonate, and barium phosphate are formed.[2] In contradistinction to the other two salts, barium chlorid is soluble in warm water. The residue is therefore extracted with hot water, filtered, and the amount of barium present in the filtrate determined either by titration with potassium bichromate or by a weighing method after conversion of all the barium into barium sulphate (von Jaksch,[3] Leo[4]). Bourget[5] has described a modification of Sjöqvist's method. Ewald,[1] according to Salkowski, uses the following modification for obtaining a general estimate of the amount of acid present. He adds a few drops of concentrated sodium carbonate solution to the filtrate of the residue just described, and estimates the amount of barium chlorid in the solution from the intensity of the cloudiness that appears.

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[1] Sjöqvist, "Eine neue Methode, freie Salzsäure im Mageninhalte quantitativ zu bestimmen," *Zeitschr. f. physiol. Chemie*, vol. xiii., p. 1.

[2] According to Kossler ("Beiträge zur Methodik der quantitativen Salzsäurebestimmung im Magen," *Zeitschr. f. physiol. Chemie*, vol. xvii., p. 91), the presence of large quantities of phosphates indicates a loss of HCl. Mizerski-Nencke (*Virchow's Jahresb.*, 1892) assert that, owing to the liberation of Ba from $BaCl_2$ and $BaCO_3$ under the influence of heat, an erroneous test, showing an increase of HCl, is obtained.

[3] Von Jaksch, *Klin. Diagnostik innerer Krankheiten*, third edition, 1892.

[4] Leo, "Beobachtungen zur Säurebestimmung im Mageninhalt," *Deutsch. med. Wochenschr.*, 1891, No. 41.

[5] Bourget, "De l'acide chlorhydrique dans le liquide stomacal," *Arch. de méd. expérimentale*, 1889, No. 6

The methods of Hayem and Winter[2] and of Martius and Lüttke[3] are also based on the process of incineration. The former method attempts to determine, first, the total quantity of chlorin; second, the amount of chlorin combined with alkali; third, the quantity of free hydrochloric acid. This method would be preferable to other methods only if it were possible to obtain exact figures for free hydrochloric acid. A number of investigations, however, by Martius and Lüttke,[4] Sansoni,[5] F. A. Hoffmann[6] have showed that this is not the case, for evaporation and drying of the stomach-contents do not cause all the free hydrochloric acid to disappear.

This method is executed as follows: Into three porcelain crucibles, *a*, *b*, and *c*, are poured 5 c.c. of filtered stomach-contents. Into *a* is poured an excess of concentrated sodium carbonate solution. The contents of all three crucibles is evaporated on the water-bath. Of the three crucibles, *b* is heated for another hour at 100° F. in order to drive off all free hydrochloric acid. Then sodium carbonate solution in excess is added, and the mixture again evaporated. The substance in crucible *c* is evaporated without any additions in order to drive off all free, and later, by glowing, all the combined, chlorin. After evaporation all three crucibles are heated to a red heat. It is well not to heat too long, as otherwise some of the inorganic chlorin combinations may become dissociated. The carbonaceous residue of *a* and *b* is extracted with hot water containing some nitric acid. The carbonic acid gas is driven off by boiling. The residue in *c* is leached out with hot water and immediately examined. The extracts are filtered through a filter containing no chlorin, and the residue washed repeatedly with hot distilled water. In the filtrate the chlorin is determined with $\frac{1}{10}$ normal silver solution, using potassium chromate as an indicator. The contents of crucible *a* informs us in regard to the total amount of chlorin present ($T =$ total chlorin). $b =$ the combined $+$ the fixed chlorin, and $c = F$ $=$ fixed chlorin; $b - c$ gives the value of combined hydrochloric acid $=$ $c =$ combined chlorin; $a - b$ gives the value for free hydrochloric acid $= \mathrm{H} =$ free HCl; A, determined by titration with $\frac{1}{10}$ normal alkali solution and phenolphthalein, indicates the total acidity.

The method of Martius and Lüttke is the one most frequently employed nowadays for clinical purposes. It is executed very much like the one that Volhard has described for determining the chlorids in the urine. The chlorin is determined after conversion into chlorid of silver. The total amount of chlorin and the quantity combined with alkali are determined; by subtracting the latter from the former, the sum of free plus combined hydrochloric acid is obtained. Martius and Lüttke, as we have already mentioned, group these two factors under the name of physiologically active hydrochloric acid. The method of Martius and Lüttke is executed as follows. The following normal solutions are necessary:

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[1] Ewald, *Klinik der Verdauungskrankheiten*, third edition, vol. ii.

[2] Hayem and Winter, *Du chimisme stomacal*, Paris, 1891.

[3] Martius and Lüttke, *Die Magensäure des Menschen*, Stuttgart, 1892.

[4] *Die Magensäure des Menschen*, Stuttgart, 1892.

[5] Sansoni, *Berlin. klin. Wochenschr.*, 1892, No. 43.

[6] F. A. Hoffmann, *Schmidt's Jahrbücher*, vol. ccxxxiii., p. 268.

1. **One-tenth normal silver solution** containing 17 gm. of silver nitrate to a liter. To the solution the indicator, sulphate of iron, is added, also a little nitric acid in excess. It is best prepared as follows: 17.5 gm. of nitrate of silver are dissolved in about 900 c.c. of nitric acid of 25 per cent.; to the solution, 50 cm. of liquor ferri sulfurici oxydati are added. The fluid is then filled up to a liter. The solution is standardized with $\frac{1}{10}$ hydrochloric acid in the customary manner.

2. **One-tenth ammonium sulphocyanid solution** containing 7.6 gm. to the liter of $CNSNH_4$; 8 gm. of ammonium sulphocyanid are dissolved in a liter of water, and the solution accurately titrated with $\frac{1}{10}$ normal silver solution. In order to do this 10 c.c. of the silver solution are poured into the beaker and about 150 to 200 c.c. of water added. The sulphocyanid solution is then allowed to drop into this mixture from a buret while the fluid is being stirred. Finally a reddish color will appear. If, for instance, 9.7 c.c. were used for this purpose, 970 c.c. of the sulphocyanid solution must be diluted to 1000 c.c. A second test is made with this solution, and in this way it is determined whether the solution is exactly $\frac{1}{10}$ normal.

On titrating with the sulphocyanid solution it will be found that the amount of silver that was not used for the formation of silver chlorid gives silver sulphocyanid until all the silver is converted. As soon as this occurs, sulphocyanid of iron is formed, and the fluid turns blood-red.

The method must be divided into two parts: on the one hand, the determination of the total chlorin (*a*); on the other, the determination of the chlorids (*b*), so that the value of hydrochloric acid is $a + b$. The details of this method are the following:

(*a*) *Determination of the Total Chlorin.*—Ten c.c. of stomach-contents, either filtered or, according to Martius, unfiltered, are poured into a graduated flask containing 100 c.c. The cylinder in which the 10 c.c. of stomach-contents are measured must be washed out once or twice with water; then 20 c.c. of a normal silver solution are added, the whole shaken, and allowed to stand for ten minutes.

If the stomach-contents is stained, 5 to 10 drops of a permanganate solution (1 : 15) may be added in order to discolor it. In the majority of cases this is not necessary, and the permanganate should never be added until all the chlorin is combined with silver; if it is added before, the permanganate will decompose the hydrochloric acid and elaborate free chlorin. This will evaporate and render the analysis doubtful. After the discoloration has been performed, the flask is filled so that it contains 100 c.c., again shaken, and filtered through a dry filter into a dry vessel. Fifty c.c. of this filtrate are then titrated with sulphocyanid solution in a beaker.

The calculation for chlorin is performed as follows: The number of cubic centimeters of sulphocyanid solution are multiplied by 2, and the figure obtained subtracted from the amount of silver (20 c.c.) employed.

(*b*) *Determination of Chlorids.*—Ten c.c. of stomach-contents are evaporated to dryness in a platinum dish on the water-bath. If a water-bath cannot be procured, a thick asbestos plate that is heated by a gas or alcohol lamp may be used. In this manner the evaporation to dryness proceeds rapidly and none of the fluid is lost by sputtering.

The dry residue is burned over a direct flame. It is well to heat only until the coal no longer burns with a luminous flame; if the mass is heated too long, some of the chlorid may evaporate. After combustion of the dry residue, the carbonaceous material remaining is powdered with a glass rod and extracted with 100 c.c. of warm water; the extract is then filtered. We know from experience that this amount of water is sufficient to extract all the carbonaceous material. If there is any doubt in the mind of the investigator whether all the chlorin has been leached out or not, a few drops of silver solution may be added to the filtrate; if chlorin is present, a white precipitate will be formed, and this will indicate that more water must be used.

The whole filtrate is then poured into a beaker, 10 c.c. of $\frac{1}{10}$ silver solution are added, and titration is performed with $\frac{1}{10}$ silver cyanid solution.

The chlorin is calculated by subtracting the number of cubic centimeters of sulphocyanid solution from the quantity of silver solution used (10 c.c.).

(*c*) *Calculation of Hydrochloric Acid* (*a* — *b*).—The amount of hydrochloric acid present in 10 c.c. of stomach-contents may be learned by subtracting the two values obtained above—namely, total chlorin (*a*) and total chlorids (*b*). The total chlorin and the chlorin in combination are both expressed in cubic centimeters of $\frac{1}{10}$ normal solution, so that the subtraction of the two gives the desired figure; by multiplying this figure with 0.0365, the absolute quantity of hydrochloric acid present in 100 c.c. of stomach-contents or the percentage value of hydrochloric acid is found.

We may add that the amount of $\frac{1}{10}$ silver solution that we employ (*a*, 20 c.c., *b*, 10 c.c.) is sufficient, according to our experience, to bind all the chlorin. If, in rare cases, stomach-contents should contain more chlorin, more nitrate of silver will have to be added (Martius).

Leo[1] has published another method that is based on altogether different principles than those enumerated. The methods of Sjöqvist and Hayem and Winter, as well as that of Martius and Lüttke, are based on incineration. The method of Leo was discovered before the method of Martius-Lüttke, and at a time when those of the other two investigators were contending for supremacy. Leo based his method on the supposition that stomach-contents in the majority of cases contains only very small quantities of organic acid when it is removed from the stomach for diagnostic purposes, and that the most important sources of error are the acid salts. He claimed, therefore, that it was only necessary, in order to determine the free plus the combined hydrochloric acid, to subtract that amount of acidity which was due to acid salts from the total acidity determined by titration with phenolphthalein. This can be done with carbonate of calcium, for this substance has the property of converting both free and combined hydrochloric acid to chlorid of calcium; this combination is a natural salt, and the reaction occurs in the cold. The acid phosphates, on the other hand, are not converted into neutral salts by calcium carbonate. The principle of the method, therefore, is the following: The total acidity is at first determined, then calcium carbonate is added, and the amount of acid due to acid phosphates determined; if the latter is subtracted from the former, the acidity due to free and combined hydrochloric acid is found, provided, of course, that no organic acids were present in the stomach-contents filtrate. If necessary, those organic acids can first be removed (shaking the stomach-contents with ether in order to remove the lactic acid), although this manipulation impairs the ultimate result.

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[1] *Loc. cit.*, and *Diagnostik der Krankheiten der Verdauungsorgane*, Berlin, 1890.

Another peculiarity of the reaction between acid phosphates and calcium chlorid must be mentioned. A given quantity of acid phosphate requires twice as much normal sodium hydrate solution for neutralization in the presence of chlorid of calcium as it does in the absence of this substance. Knowing this, we determine the total acidity and the acidity of acid phosphates under identical conditions—that is, in both cases in the presence of chlorid of calcium in excess, so that the method is performed as follows: At first the total acidity is determined by adding 5 c.c. of a concentrated chlorid of calcium solution to 10 c.c. of filtered stomach-contents, and titrating with phenolphthalein as an indicator; then a small quantity of pure carbonate of calcium is added to 15 c.c. of the stomach-contents filtrate, the mixture poured into a beaker and filtered through a dry filter into a dry vessel (beaker or pointed glass). The filtrate now contains chlorid of calcium, the acid phosphates, and free carbon dioxid. It is not practical to remove the latter by evaporation, because some of the fluid may also be evaporated and the volume of the mixture be changed; the gas must, therefore, be driven off by a current of air passed through the liquid, the current of air carrying the carbon dioxid off with it. Five c.c. of a concentrated chlorid of calcium solution are now added to 10 c.c. of the filtrate. The mixture is titrated, and the figure obtained corresponds to the acidity of the acid phosphates. If this figure is subtracted from the figure for total acidity obtained as above, the value calculated will indicate the quantity of free plus combined hydrochloric acid (of course, only in the absence of organic acid).

Quite recently Töpfer[1] has described a method that, he claims, enables us to determine the total acidity, the free hydrochloric acid, and hydrochloric acid in loose combination by titration alone. Free hydrochloric acid is determined with a 0.5 per cent. alcoholic solution of dimethylamidoazobenzol; the hydrochloric acid in loose combination is titrated with a 1 per cent. watery solution of alizarin. Strauss[1] has tested this method in my laboratory, and has found that one of the chief sources of error is our inability to determine the exact color changes in the two indicators employed. I have already mentioned that the results obtained with dimethylamidoazobenzol are not absolutely uniform.

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[1] Töpfer, "Eine Methode zur titrimetrischen Bestimmung der hauptsächlichsten Factoren der Magenacidität," *Zeitschr. f. physiol. Chemie*, vol. xix., No. 1.

The oldest method recommended is that of Mierzynski[2]; it is a modification of Sjöqvist's and the gas volumetric method. The object of this method is to enable the physician to perform quantitative determinations of hydrochloric acid without analytic weighing and standardized solutions.

The acids contained in the stomach-contents are converted into their barium salts by the addition of barium carbonate, just as in the method of Sjöqvist. The barium salts of the organic acids are then converted into barium carbonate by heating, and the amount of barium chlorid remaining is extracted with hot water. The chlorid of barium is then precipitated by the chromate of ammonia; first, however, small quantities of barium hydroxid that may have been formed by reduction from the other salt during incineration are reconverted into carbonate by a current of air. The precipitate of chromate of barium is collected on a filter and washed with a warm dilute solution of ammonia. The filter is then perforated, and the precipitate washed into the mixer of Knop-Wagner's azotometer with hot hydrochloric acid. On addition of dilute sulphuric acid and peroxid of hydrogen, oxygen is liberated. One atom of oxygen corresponds to half a molecule of hydrochloric acid in the original solution; it is possible, therefore, to calculate the amount of hydrochloric acid present from the volume of oxygen developed.

Wiener[3] has tested this method in von Jaksch's clinic, and has shown that exact results may be obtained. Wiener does not, however, agree with Mierzynski in regard to the simplicity of the method. It is true that the balance and standardized solutions are not necessary, but the method is not simple by any means, and hardly calculated to replace the ordinary methods in use.

Indications for the Quantitative Determination of Hydrochloric Acid and Its Diagnostic Significance.—Quantitative determinations of free hydrochloric acid are important chiefly when hyperacidity is suspected. Any one with a little practice will be able at least to suspect hyperacidity if the color-reactions are particularly intense, and if certain peculiarities are found in the stomach-contents. It is probably best to proceed as we do—that is, to perform quantitative estimations of the total acidity and of free hydrochloric acid in every case in which the color-reactions are positive.

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[1] *Deutsch. Arch. f. klin. Med.*, 1895, vol. lv.

[2] Von Mierzynski, "Gasvolumetrische Säurebestimmung im Mageninhalt," *Centralbl. f. innere Med.*, 1894, No. 46.

[3] Wiener, "Ueber die klinische Brauchbarkeit der gasvolumetrischen Salzsäurebestimmung im Magensafte," *Centralbl. f. innere Med.*, 1895, No. 12.

We will now discuss a group of cases that is exactly opposed to the above—namely, those in which free hydrochloric acid is absent. If a stomach cannot produce free hydrochloric acid, it is insufficient. Aside from cases of temporary insufficiency of gastric secretion, there are a large number of cases of *sub-* or *hypaciditas hydrochlorica* that may be divided into two large groups—(1) cases in which no hydrochloric acid whatever is secreted, so that the most complicated methods fail to reveal the presence of any hydrochloric acid in combination; (2) cases in which the ordinary color-reactions do not demonstrate the presence of free hydrochloric acid, but in which there is a relatively large amount of hydrochloric acid in combination. In order to determine the degree of this hydrochloric acid insufficiency quantitative determinations of hydrochloric acid in combination must be performed. Von Noorden and Honigmann (see also Biedert-Langermann[1]) have devised a method which is simple and sufficient for practical purposes. They determine the degree of hydrochloric acid insufficiency by adding $\frac{1}{10}$ normal hydrochloric acid to the stomach-contents until free hydrochloric acid can be detected by phloroglucin-vanillin or Congo-paper. The quantity of hydrochloric acid necessary to do this will indicate how much more hydrochloric acid the stomach should have secreted in each individual case; in other words, what the degree of gastric insufficiency in regard to hydrochloric acid secretion was.

The technic of this method is similar to the quantitative determination of free hydrochloric acid that we have described above. Ten c.c. of gastric contents filtrate are measured in a measuring flask; the fluid is poured into a beaker, and $\frac{1}{10}$ normal hydrochloric acid allowed to flow into it until Congo-paper shows a faint bluish tinge. It is well to control the color-change on the Congo-paper by moistening it with a drop of distilled water. The deficit of free hydrochloric acid can be determined in a similar manner by using Günzburg's reagent as an indicator. From 25 to 30 drops of the latter are added to 10 c.c. of stomach-contents, and $\frac{1}{10}$ normal hydrochloric acid allowed to flow into the mixture until a red mirror appears on a porcelain dish if a drop or two of this mixture is evaporated.

The other method of determining the degree of hydrochloric acid insufficiency is to determine the quantity of combined hydrochloric acid. In my opinion[2] there are only very few cases in which it is necessary, from a practical point of view, to do this. We have already mentioned that in cases of hyperacidity, or in any case where free hydrochloric acid is present, the quantitative determination of the total acidity and of free hydrochloric acid is much more important than the determination of combined hydrochloric acid; at all events, the former estimation is sufficient for practical purposes. In cases of subacidity the determination of combined hydrochloric acid is of subordinate practical importance, although a number of theoretically interesting and important questions may be determined by this estimation. For instance, the question whether the mucous membrane has secreted anything at all can be answered decisively only by this determination. Cases of anacidity —that is, complete cessation of all secretion of hydrochloric acid—are exceedingly rare. The question whether an atrophic process involving the gastric glands is progressing or is stationary may be determined either by an estimation of the amount of hydrochloric acid present in the gastric juice or by a determination of the hydrochloric acid deficit.

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[1] Langermann, "Untersuchungen über die quantitative Bestimmung der Salzsäure im Mageninhalt," *Inaug. Diss.*, Giessen, 1892, and *Virchow's Arch.*, vol. cxxviii.

[2] On this point consult also Honigmann, *Berlin klin. Wochenschr.*, 1893, Nos. 15, 16; von Noorden, *ibid.*, No. 19, and Ewald, *ibid.*, No. 19.

We cannot give a complete critical review of the different methods for determining the quantity of hydrochloric acid. We refer for such a critique to the work of Martius-Lüttke. We believe that the following methods are the most useful for the general practitioner : if free hydrochloric acid is present or absent, the method of Martius-Lüttke ; if free hydrochloric acid is absent, the method of von Noorden and Honigmann. We recommend the method of Martius-Lüttke, notwithstanding the fact that it has all the defects that are common to the methods of glowing the residue in the presence of ammonia compounds.[1] We think, however, that this disadvantage is compensated by the rapidity and simplicity of executing this method and by the relatively exact results that are obtained. We believe that the method of Honigmann and von Noorden is by far the best one if free hydrochloric acid is absent. It gives us a clear insight into the actual conditions, and is exceedingly easy to execute. We believe that in cases of the latter kind it is just as important to know the degree of acid insufficiency as to know how much hydrochloric acid the gastric mucosa was able to secrete.

Most authors call 0.1 to 0.2 per cent. of hydrochloric acid normal ; if more than 0.2 per cent. is present, they speak of hyperacidity. Strictly speaking, however, it is not correct to judge the amount of hydrochloric acid excretion from the total acidity, even though the color-reactions are considered at the same time.

In all cases of this kind both the total acidity and the amount of free hydrochloric acid should be determined, and even if this is done, it is impossible to give exact normal figures, as the total acidity and the free hydrochloric acid excretion fluctuate physiologically. Aside from the fact that the time at which stomach-contents is removed, after the administration of the test-meal, exercises an influence on the amount of total acidity and of free hydrochloric acid, there seem to be certain geographic differences in the secretion of acid. Strauss,[2] for instance, calculated the average total acidity of local cases in my clinic. He examined 170 cases, excluding, of course, those of carcinomata. He found the total acidity after the test-breakfast to be 68. A summary of 92 cases, on the other hand, again exclusive of those of carcinomata, studied in Berlin, showed a total acidity of only 47 after a test-breakfast. In the summary from our clinic the free hydrochloric acid value was 37, but we have repeatedly found as much as 80 to 100.

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[1] *Cf.* Rosenheim, *Centralbl. f. klin. Med.*, 1892, No. 32 ; Honigmann, *Berlin. klin Wochenschr.*, 1893, Nos. 15, 16 ; and Strauss, *ibid.*, 1893, No. 17.

[2] *Deutsch. Arch. f. klin. Med.*, 1895, vol. lv.

In this locality normal values after a test-breakfast are probably 40 to 60 for free hydrochloric acid. After a test-meal an average value of 111 for total acidity, and of 44 for free hydrochloric acid, has been found in our clinic. Here, too, we have observed values of 90 to 100 for free hydrochloric acid. Average figures, then, for total acidity are 50 to 75 ; for free hydrochloric acid, 20 to 40. Naturally, the amount of combined acid is greater after a test-meal than after a test-breakfast. These normal figures, of course, represent approximate values ; the same figure may mean hyperacidity in one individual and not in another. Cases of hyperacidity are quite frequently seen in which no high values of hydrochloric acid are found on quantitative analysis. At the same time the symptoms disappear in cases of this character as soon as antacids are administered. This indicates that in these cases there was an increase of hydrochloric acid which was only relative, but at the same time caused the symptoms. On the other hand, cases of abnormally high hydrochloric acid excretion are seen that show no symptoms of hyperacidity. This can be explained from geographic and individual differences in the physiologic excretion of hydrochloric acid. If the stomach is accustomed to perform its functions with a relatively small quantity of hydrochloric acid, a plus will cause disturbances in such a case, whereas it will not produce symptoms in another case that is physiologically accustomed to a high excretion of hydrochloric acid. It would be better, therefore, in defining hyperacidity not to designate the total acidity, but the quantity of free hydrochloric acid. There can be no such thing as hyperacidity without free hydrochloric acid ; the increase of hydrochloric acid in combination does not indicate hyperacidity, but only the absolute or relative increase of free hydrochloric acid. It is the latter that constitutes hyperacidity and causes the symptoms of this condition.

It is true, at the same time, that we cannot draw a sharp distinction between the normal and the pathologic in this respect ; nor can we indicate the normal amount by a definite figure.

[In America the presence of free hydrochloric acid above 0.2 per cent. is not often observed, and when even 0.2 per cent. of free hydrochloric acid exists, it is, in American patients, usually accompanied by symptoms of hyperacidity. Hyperesthesia to the effect of hydrochloric acid is apparently more common with us than with other people.—Ed.]

The Determination of the Absolute Quantity of Hydrochloric Acid According to Bourget and Geigel.—The methods described so far for the determination of free and combined hydrochloric acid and of the total acidity indicate the percentage values for hydrochloric acid or for the acid factors in general. Bourget,[1] and independently Geigel and Blass,[1] have devised a method for determining the total quantity of acid present in the stomach-contents at a given time; they draw their conclusions in regard to the functional powers of the stomach from this finding, and not, as other investigators, from the percentage values. Geigel's original method is the following: One hour after the administration of a test-breakfast a small quantity of undiluted stomach-contents is expressed. The stomach is then repeatedly washed out with large quantities of water until the washings are quite clear. All the washings are collected in a second vessel. The amount of fluid is about 2 liters.

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[1] Bourget, "L'acide chlorhydrique dans le liquide stomacal," *Arch. de méd. exp.*, 1889, No. 6.

The undiluted stomach-contents obtained by expression is measured and filtered. In 2 c.c. of this filtrate the percentage of hydrochloric acid is determined according to the method of Braun. The washings are then also filtered,[2] the residue repeatedly washed with distilled water, the filtrate and the washings combined, mixed, and measured. Fifty c.c. of this fluid are mixed, according to Braun's method, with $\frac{1}{10}$ normal sodium hydrate solution in excess, carefully evaporated to dryness in a platinum crucible, and finally incinerated. The ash is dissolved in $\frac{1}{10}$ normal sulphuric acid, equal in amount to the quantity of $\frac{1}{10}$ normal hydrate solution employed, the carbon dioxid removed by careful heating, and the fluid finally neutralized with $\frac{1}{10}$ normal sodium hydrate solution, using two drops of alcoholic phenolphthalein solution as an indicator; in other words, the washings are treated in the same manner by Braun's method as the undiluted stomach-contents. If the number of cubic centimeters of sodium hydrate solution employed are multiplied by 0.00365, the figure obtained will indicate the number of grams of hydrochloric acid present in 50 c.c. of the dilute stomach-contents. The absolute amount of free hydrochloric acid in grams can readily be calculated from the figures obtained in this manner.

This method takes a great deal of time, and Geigel has advised the following modification: A small quantity of undiluted stomach-contents is expressed; 300 c.c. of water are then poured into the stomach from the tube and allowed to flow out again. This is repeated several times with the same 300 c.c. of water. Finally, a specimen of this wash-water is analyzed. The specific gravity of the undiluted stomach-contents and of the stomach-contents diluted with 300 c.c. of water is determined with a pyknometer. The original volume of the stomach-contents is then calculated from the figures obtained. The percentage of free hydrochloric acid present in the undiluted stomach-contents is then calculated, and from this figure the specific gravity determined as above. The absolute quantity of hydrochloric acid may be indirectly determined. This method is scientifically interesting, but is not practical, for, as Ewald,[3] who figured with Geigel's own values, has shown, the percentage and absolute figures are frequently equal, or the same percentage figures may be obtained in very different quantities of stomach-contents. In addition, it is impossible by Geigel's method to obtain any information in regard to the hydrochloric acid secreted from the time that the food enters the stomach until the stomach-contents is removed. This is due to the fact that this method enables us to determine only the amount of hydrochloric acid present at the moment when the stomach-contents is removed, and the quantity of material present in the stomach is chiefly dependent on the motility of the organ, although other factors also play a rôle. Ewald calls attention to the fact that the absolute values may be changed at any time by the passage of stomach-contents into the intestine, whereas the percentage values remain unchanged.

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[1] Geigel and Blass, "Procentuale und absolute Acidität des Magensaftes," *Zeitschr. f. klin. Med.*, vol. xx.; Geigel and Abend, "Die Salzsäuresecretion bei Dyspepsia nervosa," *Virchow's Arch.*, 1892, vol. cxxx.

[2] In later experiments Geigel employed unfiltered material.

[3] Ewald, "Ueber Stricturen der Speiseröhre und einen Fall von Ulcus œsophagi pepticum, etc.," *Zeitschr. f. klin. Med.*, vol. xx.

However interesting, therefore, the determination of the absolute amount of hydrochloric acid present may be, the values obtained by this method do not give any more practical information than the simple percentage determination of hydrochloric acid.

DETERMINATION OF ORGANIC ACIDS.

Organic acids in appreciable quantities are normally never present in the stomach-contents after the administration of a test-breakfast or a test-meal. If large quantities of organic acids are present in the stomach-contents, this is a sign of abnormal fermentation, unless large quantities of organic acids have been ingested with the food. It is necessary to distinguish between these imported preformed organic acids and those that are formed in the stomach by fermentation. The latter alone are pathologically significant.

The lactic acids, notably the racemoid form, are the most important of these organic acids; sarcolactic, on the other hand, which may enter the stomach with the food, is not important. Butyric acid, acetic acid, and the volatile fatty acids are of only secondary importance. We will not discuss the physiologic occurrence of lactic acid during the early stages of digestion; we will only mention that until quite recently two or three stages of digestion were distinguished, a first stage in which only lactic acid, but no free hydrochloric acid, is demonstrable in the stomach-contents; a second one in which both lactic acid and hydrochloric acid are present, and a third one in which hydrochloric acid is alone present. This view has been refuted by the investigations of Martius and Lüttke,[1] for these authors showed that no other acids besides hydrochloric acid are concerned to any great degree in the normal process of digestion. We are justified, however, in distinguishing two stages of digestion—namely, an amylolytic stage, in which no free hydrochloric acid is present, and a proteolytic stage, in which free hydrochloric acid is seen.

If food that contains no trace of lactic acid—for instance, oatmeal gruel (Boas[2])—is eaten, no lactic acid is normally produced in any stage of the digestion of this food. Although Miller has shown that lactic acid bacilli are always present in the saliva, the formation of lactic acid does not always occur in a normal stomach, for the reason chiefly that here, aside from the bactericidal action of hydrochloric acid, the food is forced onward into the intestine long before lactic acid can be generated.

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[1] *Die Magensäure des Menschen*, Stuttgart, 1892.

[2] *Zeitschr. f. klin. Med.*, vol. xxv., Nos. 3, 4.

As the only natural and correct method of making a chemical examination of stomach-contents is to analyze gastric fluid obtained by siphoning the stomach at a certain time after the administration of a test-meal or a test-breakfast, we will study only those organic acids that are found under these conditions. When a test-breakfast is taken, only minimal traces of organic acids, particularly lactic, are introduced; the same applies to the test-meal, in which some sarcolactic acid is always present; consequently the examination of the stomach-contents will never reveal the presence of organic acids (lactic acids) if the stomach is normal. For this reason the occurrence of large quantities of organic acids, particularly lactic, after the administration of a test-breakfast is always pathologic.

Occurrence and Determination of Lactic Acid.—Practically, the determination of lactic acid is the most important one, so that the general practitioner, as a rule, limits his analysis for organic acids to the determination of lactic acid. We possess a number of methods to do this, which are all more or less exact, and it appears that the degree of exactness is proportionate to the difficulty of executing the different methods.

Only those methods that are easy are of value to the practitioner, provided, of course, that they can positively determine the presence of lactic acid in pathologic quantities, even though they may be insufficient for the discovery of small traces. We will omit the more complicated methods, and limit ourselves to the simpler ones. The former consist in making lactates (zinc, lead, calcium) and studying them from a crystallographic point of view. Of the latter, Uffelmann's reaction is the most frequently employed, and the one that gives sufficiently accurate results if all necessary precautions are carefully observed. This method, it is true, has been made the subject of much adverse criticism within the last few years.

Uffelmann's reagent should be fresh, and should be prepared before each test. Uffelmann[1] recommends a mixture of 10 c.c. of a 4 per cent. carbolic acid solution and 20 c.c. of distilled water; to this he adds one drop of the official liquor ferri sesquichlorati. This mixture is a clear, amethyst-blue fluid. The following mixture is of the same color, and just as sensitive—namely, 20 c.c. of distilled water, 10 c.c. of 4 per cent. carbolic acid, and 0.1 c.c. of neutral 10 per cent. iron chlorid solution; a very dilute solution of iron chlorid alone—that is, one drop of liquor in 50 c.c. of distilled water—is also sufficient. The solution may also be prepared by diluting one drop of the official chlorid of iron solution with distilled water until the solution is almost colorless, and then adding a 2 to 4 per cent. solution of carbolic acid until a transparent amethyst-blue color appears. As a rule, a few drops of carbolic acid solution are sufficient. The blue color of the iron chlorid solution is simply a contrast color, for the reaction proper occurs by contact of lactic acid with dilute iron chlorid solution. The disappearance of the blue color merely indicates the beginning of the reaction.

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[1] Uffelmann, "Ueber die Methode der Untersuchung des Mageninhaltes auf freie Säuren," experiments on a gastrotomized subject, *Deutsch. Arch. f. klin. Med.*, vol. xxvi., 1880, and Uffelmann, "Ueber die Methoden des Nachweises freier Säuren im Mageninhalt," *Zeitschr. f. klin. Med.*, vol. viii., p. 392.

We say the reaction is positive if the amethyst-blue color of the iron chlorid carbolic acid solution turns yellow or yellowish green on addition of stomach-contents filtrate. Formerly this shade was called canary yellow; modern authors prefer to call it canary green. It is important to obtain a distinct mental impression of this color, as it is difficult to describe.

In doubtful cases it is well to control the reaction with a dilute lactic acid solution. The color obtained in this manner is compared with that obtained with the stomach-contents.

The following plan is a practical one: The reagent is prepared and poured into three test-tubes that are placed side by side; into one is poured stomach-contents; into another, the same quantity of dilute lactic acid solution; into the third, the same quantity of distilled water or dilute hydrochloric acid solution. Only if a distinct yellow or yellowish-green color appears is the presence of lactic acid demonstrated. This reagent is sensitive both to lactic acid and to lactic acid salts.

A number of other substances are capable of producing the same or a similar discoloration of dilute iron chlorid solutions. Among these we may mention oxalic acid, citric acid, tartaric acid, alcohol, dextrose, phosphates, etc. All these bodies, however, are never found in the filtrate of stomach-contents if all the precautions are taken that we postulate; if they do occur, conditions exist at the same time that prevent a positive Uffelmann reaction. For this reason a positive reaction, provided all necessary precautions are taken, is, in my opinion, still of diagnostic value. Only large quantities of lactic acid are practically significant, and such large quantities can always, to judge from our own observations, be demonstrated by Uffelmann's reagent. Even Boas,[1] to whom we owe the introduction of more delicate and more exact methods, says that in those cases in which a large amount of lactic acid could be determined by his own method, which we will describe below, Uffelmann's reaction was also positive after a test-breakfast. Rosenheim[2] says the same; he claims that Boas's reaction is never positive where Uffelmann's reaction is not also distinctly positive. In our clinic similar observations have been made, so that we feel justified in advocating the clinical employment of Uffelmann's reagent under the conditions outlined. We place ourselves in opposition in this respect to Martius[1] and Penzoldt.[2]

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[1] Boas, "Eine neue Methode der qualitativen und quantitativen Milchsäurebestimmung im Mageninhalt," *Deutsch. med. Wochenschr.*, 1893, No. 39; see also *Münch. med. Wochenschr.*, 1893, No. 43.

[2] Rosenheim, "Ueber einen Fall von Gastritis gravis," *Berlin. klin. Wochenschr.*, 1894, No. 39.

Uffelmann's reaction has several sources of error. For this reason a number of modifications of the method have been recommended. One of the best known of these methods is adding to stomach-contents ten volumes of ether, shaking the mixture, evaporating the ether, and dissolving the residue in water. Uffelmann's reaction is then performed with this watery solution. Fleischer[3] does not evaporate the ether, but adds fresh iron chlorid carbolic acid solution directly to the ether. If lactic acid is present, the watery solution will be stained yellow and appear at the bottom of the test-tube.

The method devised by Kelling[4] is a very sensitive one. This author dilutes the filtrate of stomach-contents to 10 or 20 volumes, and to this diluted stomach-contents he adds one or two drops of a 5 per cent. iron chlorid solution. If a greenish color appears, he claims that this proves the presence of lactic acid, because lactic acid even in dilutions of 1 : 10,000 or 1 : 15,000 colors the solution greenish. Kelling attempts to eliminate the brown or reddish-brown discoloration of Uffelmann's reaction, which is frequently caused by the sulphocyanid from the saliva, by adding a few drops of corrosive sublimate. In this manner he says the canary-greenish color caused by lactic acid appears, notwithstanding the presence of sulphocyanid. My former assistant, Strauss,[5] has also devised a very useful modification that has been found of value in our clinic.

For exact determinations it is important to mix equal parts of stomach-contents and of iron chlorid. In order to do this, Strauss employs a mixing funnel with two markings (see Fig. 12), one of which indicates 5 c.c., the other one, 25 c.c. The mixing funnel is filled to 5 with stomach filtrate; ether is then poured in to 25, and the whole is well shaken; then the lower cock is opened, the fluid is allowed to run off until it reaches 5, and distilled water is then poured in to 25. To this mixture are added 2 drops of an iron chlorid solution (1 : 9 distilled water), and the whole shaken. Comparative investigations by Strauss have shown that if 1 pro mille of lactic acid is present, an intense green color appears; if there is less lactic acid, the color is light green. One of the advantages of this method is the absence of a milky clouding.

Boas[6] recommends oxidation of lactic acid to acetaldehyd and formic acid. The presence of aldehyd is demonstrated by the iodoform reaction with alkaline iodin solution, or by the formation of aldehyd mercury with Nessler's reagent. (For the details of this method, see below.) The best method for making quantitative determination of lactic acid is to manufacture the zinc or lead salt and to determine the amount formed by analytic weighing. Another good method is the transformation of lactic acid into aldehyd according to Boas' method, followed by titrimetric estimation of the amount of iodoform formed from aldehyd. There are other methods that are less exact and cannot be employed for quantitative determinations. Among these may be mentioned the following two: The quantity of lactic acid is determined by shaking the stomach-contents with ether; the ether is evaporated, and the acidity of the residue dissolved in water determined, or again the acidity of the stomach-contents is determined before and after the extraction with ether. For scientific purposes Boas's method is the one universally employed. Boas determines lactic acid quantitatively as follows:

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[1] Martius and Lüttke, *Die Magensäure des Menschen*, Stuttgart, 1892, p. 56.

[2] Penzoldt, "Beiträge zur Lehre der menschlichen Magenverdauung," *Deutsch. Arch. f. klin. Med.*, vol. liii.

[3] Quoted from Penzoldt, *ibid.*, vol. li., p. 544.

[4] Kelling, "Ueber Rhodan im Mageninhalt, zugleich ein Beitrag zum Uffelmann'schen Milchsäurereagens und zur Prüfung auf Fettsäuren," *Zeitschr. f. physiol. Chemie*, 1893, vol. xviii.

[5] *Berlin. klin. Wochenschr.*, 1895, No. 37.

[6] Boas, "Eine neue Methode der qualitativen und quantitativen Milchsäurebestimmung im Mageninhalt," *Deutsch. med. Wochenschr.*, 1893, No. 39; see also *Münch. med. Wochenschr.*, 1893, No. 43.

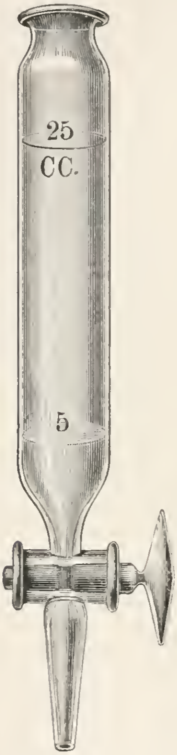

FIG. 12.—Mixing funnel.

From 10 to 20 c.c. of stomach-contents filtrate are evaporated to a syrupy consistency on the water-bath. If free hydrochloric acid is absent, the filtrate is evaporated without any additions; if hydrochloric acid is present, an excess of barium carbonate is added. The syrup is then mixed with a few drops of phosphoric acid, the carbon dioxid that is formed is driven off by boiling, the fluid is allowed to cool, and repeatedly extracted with small quantities of ether (two or three times with 50 c.c.). After half an hour the clear ethereal layer is decanted, the ether evaporated, and the residue washed into a flask with 45 c.c. of water, thoroughly shaken, and finally filtered. To the filtrate are added 5 c.c. of concentrated sulphuric acid (specific gravity 1.84) and a small quantity of manganese dioxid. The flask containing the liquid is closed with a perforated cork. Through the hole passes a glass tube that is bent and has a long limb that dips into a narrow cylinder containing 5 to 10 c.c. of an alkaline iodin solution, or the same quantity of Nessler's reagent. If the liquid is heated over a small flame, aldehyd distills into the cylinder if lactic acid is present. In this case the iodoform reaction (clouding and odor of iodoform) or, if Nessler's reagent is used, yellowish-red aldehyd mercury, appears.

This method of Boas is sufficiently accurate to enable us to perform exact quantitative determinations of lactic acid. This determination is executed exactly as above, with this difference, however, that the residue after the evaporation of ether is mixed with manganese dioxid and sulphuric acid and carefully distilled with sufficient cooling. The distilling flask is closed with a stopper containing two holes. From one of these a bent glass tube leads to the cooler; through the other passes another glass tube that is also bent, and terminates in a small piece of rubber tubing with a clamp. The purpose of the latter tube is the following: After the distillation is over all traces of aldehyd present in the flask and the cooler can be driven into the reagent by a current of air. The distillation should be carried on until about four-fifths of the fluid has distilled over. Lactic acid is now quantitatively determined by simple titration. The following solutions are necessary for this titration:

1. One-tenth normal iodin solution.
2. One-tenth normal sodium arsenite solution, or, instead, $\frac{1}{10}$ thiosulphate solution.
3. Hydrochloric acid of the specific gravity of 1.018.
4. Potassium hydrate solution (about 56 gm. of potassium hydrate to a liter of water).
5. Dilute fresh starch solution.

The distillate is poured into a high Erlenmeyer flask, and about 10 to 20 c.c. of $\frac{1}{10}$ iodin dissolved in 20 c.c. of potassium hydrate solution of the above concentration added, the mixture thoroughly shaken and allowed to stand, well corked, for several minutes. Then 20 c.c. of hydrochloric acid (1.018) are added; further, sodium bicarbonate solution in excess. Then the solution is completely discolored with a sufficient quantity of $\frac{1}{10}$ sodium arsenite solution that must correspond exactly to the $\frac{1}{10}$ iodin solution; then fresh starch solution is added, and the mixture titrated back until a blue color appears. The number of cubic centimeters of $\frac{1}{10}$ arsenious acid used are subtracted from the number of cubic centimeters of the $\frac{1}{10}$ iodin solution that have been employed, and the difference indicates the quantity of iodin that was used for the formation of iodoform; from this the quantity of aldehyd or of lactic acid can be determined.

One c.c. of $\frac{1}{10}$ iodin solution corresponds to 0.003388 gm. of lactic acid. All that is needed, therefore, is to multiply the number of cubic centimeters of $\frac{1}{10}$ iodin with this number.

In view of the fact that nearly all articles of food contain more or less of preformed lactic acid, Boas has recommended a test-meal containing absolutely no lactic acid. This consists of a tablespoonful of Knorr's oatmeal with a little salt and one liter of water. He allows this test-meal, if we understand his different publications correctly, to remain in the stomach for varying periods of time. He states that the best method is to administer this gruel in the evening, after thoroughly cleansing the stomach, and to pump out the residue the next morning.

There are a number of objections to the universal application of Boas' method for determining lactic acid in practice. In the first place the author himself states—and other authors agree with him—that in those cases where there is much lactic acid Uffelmann's reaction with a test-breakfast is also positive and sufficient for all practical purposes. We can corroborate this statement on the basis of our own experiments, and might add that if the necessary precautions are observed, Uffelmann's reaction is altogether sufficient for any practical purpose.

In the second place a new test-meal complicates our diagnosis and makes it very difficult for different authors to agree on what they mean when they describe the results of a chemical examination of the stomach-contents. Those factors that must be determined in order to obtain values that can be compared in the case of Boas' lactic acid test-meal are manifold, and it seems that the quantity of this test-meal ingested and the time[1] that it is allowed to remain in the stomach vary in different cases so much that it is almost impossible to perform the experiment under identical conditions; in addition, it is very difficult, to judge from our personal experience, to procure ether that is free from substance that can form aldehyd. Seelig[2] records the same experience. This writer was obliged to remove all aldehyd-forming substances from so-called "alcohol-free" ether by a very complicated method. After he had done this, Nessler's reagent gave a slight and gradually increasing cloudiness after two minutes.

At all events this method, however interesting and valuable it may be from the scientific point of view, is not suitable for practice, and as Boas himself insists that only the presence of large quantities of lactic acid is important from a clinical and diagnostic point of view, those slight quantities of lactic acid that are present, for instance, in the test-meal are of no significance, and we can say again that Uffelmann's reaction is sufficient for all practical purposes.

In regard to the diagnostic significance of the presence of lactic acid we may say that it indicates the existence of two conditions at the same time—namely, subacidity and stagnation. Boas[3] has claimed that stagnation and deficiency of hydrochloric acid may be present, and that, nevertheless, the production of lactic acid need not occur after a carbohydrate meal, but that a third factor was necessary for this. He regarded the appearance of large quantities of lactic acid as specific for carcinoma of the stomach.

We have been in the habit of examining all stomach-contents for lactic acid ever since Uffelmann first published the results of his investigations some fifteen years ago, and we can agree with Boas that large quantities of lactic acid are very frequently found in carcinoma of the stomach; we can go further and say that only in exceptional cases of this disease is lactic acid absent. In addition, we know that it is very rarely found in other diseases of the stomach. We believe, however, that the genesis of lactic acid under these conditions is due to deficiency of acid in carcinoma, as this deficiency allows the development of ferment organisms. We agree in this respect with Ewald,[1] Rosenheim,[2] Klemperer,[3] Strauss,[4] and others.

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[1] We have repeatedly seen from one-half to one liter of farinaceous soup disappear from the stomach within an hour to an hour and a half, so that at the expiration of this time nothing could be aspirated for purposes of analysis—this in cases that were unquestionably carcinoma.

[2] Seelig, "Die diagnostische Bedeutung der Milchsäurebestimmung nach Boas," *Berlin. klin. Wochenschr.*, 1895, No. 5.

[3] Boas, "Ueber das Vorkommen von Milchsäure im gesunden und kranken Magen, nebst Bemerkungen zur Klinik des Magencarcinoms," *Zeitschr. f. klin. Med.*, vol. xxv., Nos. 3, 4.

The investigations of Strauss in particular show the significance of acid deficiency and stagnation in the development of lactic acid. This author also shows that this obtains even if the motility of the stomach is good. He illustrates his position by a case that occurred in our clinic. It is possible that the following quotation correctly explains the tenacity with which ferment organisms may cling to the stomach-walls even though coarse morsels of food are pushed onward: "Carcinoma forms a solid plate in the stomach-walls and causes induration of the surrounding tissues. The peristaltic movements of the stomach do not exercise the same effect on this resisting hard surface that they do on the smooth mucous membrane. In the latter tissue, mountain and valley alternate, consequently the membrane is continuously cleansed."

If the stomach is healthy, the oatmeal is propelled onward and contact with saliva is not sufficiently long to allow the formation of lactic acid. If the motor power of the stomach is reduced, the same favorable conditions for the formation of lactic acid are created as in reduction of the secretory powers. Both conditions are very frequently found in carcinoma—more frequently in this lesion than in any other disease of the stomach. At the same time we fail to see anything specific for carcinoma in the occurrence of lactic acid in the stomach. A large number of observations have been chronicled in the mean time in which lactic acid was absent despite the presence of cancer of the stomach. Klemperer[5] has recently reported 3 such cases in which the diagnosis was corroborated by autopsy. On the other hand, many reports[6] are being published of large lactic acid formation in the stomach in the absence of carcinoma. These are cases of insufficient acid secretion and of stagnation. Strauss[7] has reported from our clinic a case in fat-necrosis of the pancreas, and we have observed a case with a great amount of lactic acid that proved to be an invagination of the colon and a part of the duodenum. We have also found lactic acid in 2 cases of regurgitation of intestinal contents.

According to our view, which corresponds with that of most authors who have investigated the lactic acid question within recent years, the presence of lactic acid in the stomach-contents simply indicates the existence of subacidity and of stagnation.[1] These two conditions are never so constantly present nor so intense as in carcinoma.

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[1] Ewald, *Verein f. innere Med.*, 1894; discussion of Rosenheim's paper on "Gastritis gravis."

[2] *Berlin. klin. Wochenschr.*, 1894, No. 39; *Deutsch. med. Wochenschr.*, 1895, No. 15.

[3] Klemperer, *Verhandl. des Vereins f. innere Med.*, 1895; *Deutsch. med. Wochenschr.*, 1895, No. 14.

[4] Strauss, "Ueber Magengährungen und deren diagnostische Bedeutung," *Zeitschr. f. klin. Med.*, vol. xxvi., xxvii.

[5] *Deutsch. med. Wochenschr.*, 1895, No. 14.

[6] *Cf.* Bial, "Milchsäurebildung im Magensaft bei Ulcus ventriculi mit Gastritis atrophicans und Gastrektasie," *Berlin. klin. Wochenschr.*, 1895, No. 6, and Rosenheim, *Deutsch. med. Wochenschr.*, 1895, No. 15.

[7] *Loc. cit.*

The discovery of lactic acid in the stomach-contents is, therefore, a valuable adjuvant to diagnosis. At the same time lactic acid is no pathognomonic symptom of cancer. The anatomic process does not determine the character of gastric fermentation, but determines, in the first place, the amount of hydrochloric acid secretion; in the second place, the motility of the stomach; and in the third place, the medium on which the germs can grow. We have failed to find lactic acid in a number of cases of carcinoma in which free hydrochloric acid was absent, but the motility of the stomach was normal.

The determination of other organic acids has so far never proved of practical diagnostic value. If these organic acids were not ingested they, also, indicate the existence of fermentative processes favored by stagnation. As a rule, gastric insufficiency is a necessary condition for the development of such fermentation, although, for instance, butyric fermentation may occur even in the presence of large quantities of hydrochloric acid. Butyric acid and the higher aliphatic acids may be revealed by their odor, or the filtered stomach-contents may be heated in a test-tube and the vapors allowed to pass over a piece of moistened blue litmus-paper. If volatile acids are present, the escaping vapors will color the paper red. If large quantities of filtered stomach-contents can be obtained, the filtrate may be divided and distilled. This procedure, however, is not an accurate one, because decomposition may occur. If this distillation is performed, the reaction and the acidity of the distillate may be tested.

It is hardly necessary, for practical purposes, to identify any of the organic acids other than lactic that are formed in the stomach. Acetic acid, after careful neutralization, gives a blood-red color on addition of one or two drops of chlorid of iron solution. This is due to the formation of acetate of iron. Butyric acid has the peculiar property of separating in the form of a small drop of oil on addition of small pieces of chlorid of calcium.

Titration of the total quantity of the organic acids should be performed in the same manner as the quantitative determination of hydrochloric acid; the total acidity is determined and the hydrochloric acid subtracted, the difference expressing the amount of organic acids present, excepting the organic acid salts.

GAS FERMENTATION.

Gas fermentation in the stomach has been described for many years (Wilson, Graham-Jenner, Hardwick-Budd, Carius, Friedreich, Waldenburg, Friedreich-Schulzen, Popoff, Ewald-Ruppstein, McNaught). Naunyn[1] and Minkowski[2] experimented extensively on the subject during the last decade but one. The true diagnostic significance of this phenomenon, however, has been revealed only by the investigations of the last few years. Hoppe-Seyler[3] and my former assistants, Kuhn[4] and Strauss,[5] have given particular attention to this question within recent years. For a long time I have called attention to the semiotic significance of the separation of stomach-contents into three layers, and the investigations of the last-named authors have supported my views by demonstrating that gas-formation occurs particularly if stagnation of gastric contents obtains. It may, of course, happen that the separation into three layers is not caused by gas-bubbles that rise to the top, but by fat-droplets. Strauss[6] demonstrated this in a case he reported not long ago. Cases of this character, however, are rare exceptions, and certainly not the rule.

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[1] Strauss and Bialocour (*Zeitschr. f. klin. Med.*, vol. xxviii.) performed a number of experiments in my laboratory to show that the formation of lactic acid stops, or rather does not occur, as soon as the fermentative action of the salivary ferment stops. It is known that the diastatic action of saliva stops in the presence of about 0.12 per cent. of HCl—*i. e.*, of HCl in general, not only of free HCl. At the same time that the sugar-forming function of the saliva in the stomach ceases, the lactic-acid-forming function also ceases.

That the presence of free hydrochloric acid in the stomach-contents does not prevent the abundant development of gas is a well-established clinical fact. Kuhn and Strauss have of late strengthened this view by their experiments. They showed that hydrochloric acid under conditions similar to those that obtain in the gastric juice possesses no disinfecting properties and does not prevent the development of yeast-cells. Solutions of hydrochloric acid and artificial gastric juice, on the other hand, do possess this property. They found, moreover, that if there was stagnation, those cases seemed to favor the development of gaseous fermentation that showed normal or excessive values for hydrochloric acid. This finding corresponds with the view advocated by me long ago. Strauss at the same time emphasizes the fact that gaseous fermentation may occur on a soil that is non-acid, together with lactic-acid fermentation. We have repeated some of these experiments and can corroborate his statements. The most important factor in the genesis of gaseous fermentation is the retention of organized ferments. Under normal conditions these organisms are not retained in the stomach, or remain there for so short a time that no fermentation occurs; whereas in cases where ferment organisms are retained, more or less abundant development of gas occurs within a comparatively short time.

It is *scientifically* interesting to determine the character and the quantity of the gases formed, also to determine the differences between the gases formed in the stomach and outside of the stomach in the incubator. From a *practical* point of view it is sufficient to know whether fresh stomach-contents contains a number of ferment organisms sufficient to produce gaseous fermentation in a suitable medium. A comparative study of the quantity of gas formed and the time necessary for its development furnishes valuable criteria.

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[1] Naunyn, "Verhältniss der Magengährungen zur mechanischen Insufficienz," *Deutsch. Arch. f. klin. Med.*, 1882, vol. xxxi.

[2] *Ibid.*, *Mittheilungen aus der med. Klinik zu Königsberg*, Leipsic, 1888.

[3] Hoppe-Seyler, "Untersuchungen über Magengährung," *Prager med. Wochenschr.*, 1892, No. 19; *Deutsch. Arch. f. klin. Med.*, vol. l.; *Verhandl. d. XI. Cong. f. innere Med.*, 1892.

[4] Kuhn, "Ueber Hefegährung und Bildung brennbarer Gase im menschlichen Magen," *Zeitschr. f. klin. Med.*, vol. xxi.; *Deutsch. med. Wochenschr.*, 1892, No. 49; 1893, No. 15.

[5] Strauss, "Ueber Magengährungen und deren diagnostiche Bedeutung," *Zeitschr. f. klin. Med.*, vol. xxvi., xxvii.

[6] *Ibid.*, "Zur Frage des chronischen Magensaftflusses nebst einigen Bemerkungen zur Frage des dreischichtigen Erbrechens und der Gasgährungen im Magen," *Berlin. klin. Wochenschr.*, 1894, No. 41.

The following procedure has been employed in our clinic for a long time; it is an easy one, and sufficient for all practical purposes.

A tube is filled with thoroughly mixed unfiltered stomach-contents and placed in the incubator at 37° C. (98.6° F.). The best form of tube to use for this purpose is that employed for the determination of sugar in the urine by fermentation (so-called Fiebig tubes); or the apparatus of Moritz[1] may be used if a suitable fermenting apparatus cannot be obtained; this apparatus consists of a test-tube closed by a rubber cork, through which passes a bent glass tube.

The test-tube is completely filled with stomach-contents and closed with the rubber stopper holding the glass tube. On pushing the cork into the test-tube some stomach-contents enters the glass tube, so that the apparatus does not contain any air. It is now inverted in a beaker. This apparatus is very simple and can easily be cleaned. It is not difficult to pour unfiltered stomach-contents into the tube, even if it contains much mucus.

Occasionally stomach-contents containing many ferment organisms does not contain any more sugar, for the reason that it has all been destroyed by fermentation. It is well, therefore, to fill a control tube with stomach-contents and to add a small quantity of powdered dextrose. If there is not enough stomach-contents to do this, it is best to add dextrose at once, for the reason that we are never sure that the stomach-contents will contain a sufficient amount of pabulum for the ferment organism, although, as a rule, it contains some dextrose. If no incubator is convenient, the tubes can be placed in some uniformly warm place, where the temperature remains between 20° and 40° C. (68°–104° F.). If no development of gas is noticed after twenty-four hours, the ferment tubes should be allowed to stand for three or four days more. This is necessary, because gaseous fermentation sometimes does not occur until several days have elapsed, or because there may be what is called protracted fermentation. After the experiment is concluded, it is best to boil the tubes and the other paraphernalia used.

It is hardly necessary, for practical purposes, to identify the different gases developed; they may be acetylene, methane, carbon dioxid, hydrogen, oxygen, nitrogen. It is an easy matter to determine the presence of carbon dioxid by allowing a small quantity of KOH to flow from a pipet to the bottom of the column of gas; the carbon dioxid is absorbed by the KOH, and the fluid moves upward to take the place of the absorbed gas.

It is also an easy matter to determine whether these gases are combustible. Hoppe-Seyler and Kuhn have shown that this is frequently the case. These investigators have reported some exact gas analyses.

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[1] Moritz, "Glykosurie und Diabetes," *Münch. med. Wochenschr.*, 1891, Nos. 1, 2.

The experiment for determining the existence of gaseous fermentation, as outlined above, is easily carried out. Either the expressed test-meal or test-breakfast or the vomit may be used. The experiments are also frequently valuable for determining whether stagnation exists or not. In our clinic we carry these tests out systematically, and in all cases where we can demonstrate gaseous fermentation we assume that there is stagnation, and this condition is usually identical with mechanical or motor insufficiency. It is true that there may be stagnation, and the result may still be negative ; or, on the other hand, that there may be a very slight development of gas, although the motor power is normal in regard to larger morsels of food. This is usually due to the fact that the ferment organisms are retained within the folds and fissures of the gastric mucosa. If fermentation is excessive, we can always assume motor insufficiency.

If the contents of the ferment-tube undergoes very rapid fermentation (a few hours), pyloric stenosis may be suspected, because this condition produces the most severe degrees of motor insufficiency. If, at the same time, the stomach-contents contains much lactic acid and no free hydrochloric acid, we may assume that a carcinoma of the pylorus causes this stenosis. Gaseous fermentation, however, is usually more intense and is more frequently seen in cases of motor insufficiency in which free hydrochloric acid is present. Gaseous fermentation can, however, occur in any form of stomach-disease in which the secretion of gastric juice is disturbed. Lactic-acid fermentation, on the other hand, can occur only if the stomach-contents is subacid.

Lactic-acid fermentation and gaseous fermentation are not produced by any one specific form of micro-organism, but both forms of fermentation may be caused by a variety of different germs, or the same germ may produce different decomposition products on a different nidus. The best-known and most carefully studied organisms are the lactic-acid bacterium of Oppler, on the one hand, and yeast, on the other.

OTHER ABNORMAL PRODUCTS OF FERMENTATION AND PUTREFACTION FORMED IN THE STOMACH.

The following products of fermentation must be considered : (1) Alcohol ; (2) different hydrocarbons (methane, ethylene) ; (3) sulphureted hydrogen.

Kuhn,[1] Hoppe-Seyler,[2] and Strauss,[3] in some recent investigations, have called attention to the presence of these substances. Ewald[4] and Ruppstein before this time reported a well-known case in which the same finding could be made. It is an exceedingly difficult and complicated matter to determine the presence of these bodies; and even though careful chemical analysis reveals their presence in the gases of the stomach, we can never know whether they were formed in the stomach or whether they were generated in some article of food that was undergoing fermentative changes prior to its ingestion. In cases of motor insufficiency this applies with particular force. The presence of alcohol can be demonstrated by Lieben's iodoform reaction; the presence of sulphureted hydrogen, by the formation of sulphid of lead; what has been said of the gases of the stomach applies with equal force to alcohol, as it is often impossible to determine whether it was formed in the stomach or in the food prior to its entrance into the stomach.

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[1] Kuhn, *Zeitschr. f. klin. Med.*, vol. xxi., and *Deutsch. med. Wochenschr.*, 1892, No. 49.

[2] Hoppe-Seyler, *Prager med. Wochenschr.*, 1892, No. 19 ; *Deutsch. Arch. f. klin. Med.*, 1893, No. 50.

[3] Strauss, *Zeitschr. f. klin. Med.*, vol. xxvi., xxvii.

[4] Ewald, "Ueber Magengährung und Bildung von Magengasen mit gelb brennender Flamme," *Reichert's and du Bois-Reymond's Arch.*, 1874.

Boas[1] succeeded in demonstrating sulphureted hydrogen in a number of severe cases of motor insufficiency; here free hydrochloric acid was present; Zawadzki[2] recently showed the same in 4 other cases of motor insufficiency. According to Boas,[3] H_2S is quite frequently found in the stomach in benign cases of gastric ectasy, whereas it is only rarely found in cases of ectasy that are due to carcinoma. If there was lactic-acid fermentation, Boas never succeeded in finding sulphureted hydrogen, and vice versâ. It is true that in some of Boas's cases the sulphureted hydrogen may have been formed from the reduction of sulphates.

The question of the formation of H_2S in the stomach calls for further investigation. I have performed some experiments in this direction, but failed to obtain any positive results.

The Examination for Pepsin.—The examination of the stomach-contents for pepsin should naturally follow the examination for HCl. The diagnostic significance of pepsin determinations is not, however, so important as the determination of HCl. We have already stated that in all cases in which there is a sufficiency of HCl there is also usually a sufficiency of pepsin—in fact, pepsin is not uncommonly present even if HCl is absent. The pepsin-forming function of the stomach seems to be more constant and more resistant to external influences than the hydrochloric-acid function of the stomach. In those cases in which free hydrochloric acid is present, the macroscopic examination of the stomach-contents after a test-meal will demonstrate whether pepsin excretion is sufficient, for absence of pepsin causes disturbances of albumin digestion. We have called attention to this in discussing the relative lack of pepsin in hyperacidity.

For all these reasons exact determinations of pepsin are, as a rule, unnecessary, and are carried out only in very severe diseases of the stomach that are complicated by the absence of hydrochloric acid—as, for instance, anadenia ventriculi, atrophic catarrh of the stomach, etc.

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[1] Boas, "Ueber das Vorkommen von Schwefelwasserstoff im Magen," *Deutsch. med. Wochenschr.*, 1892, No. 49.

[2] Zawadzki, "Schwefelwasserstoff im erweiterten Magen," *Centralbl. f. innere Med.*, 1894, No. 50.

[3] Boas, "Ueber Schwefelwasserstoffbildung bei Magenkrankheiten," *ibid.*, 1895, No. 3.

Practitioners in general, therefore, omit the direct examination for pepsin or pepsinogen, and perform this analysis only in exceptional cases. It would be more rational to examine the stomach-contents both for its hydrochloric acid and its pepsin in every case of stomach-disease.

The property of pepsin or of its precursor, pepsinogen, of converting coagulated albumin into soluble albumoses in the presence of hydrochloric acid is used in testing for pepsin—in other words, the artificial digestion of albumin is carried out. If the stomach-contents contains free hydrochloric acid, and if it digests albumin, the presence of pepsin is demonstrated; if the stomach-contents does not contain free hydrochloric acid, but if it still digests albumin after having been acidified with a sufficient quantity of hydrochloric acid, this also demonstrates that sufficient pepsin is present.

The digestion test is carried out as follows: About 10 c.c. of filtered stomach-contents are poured into a test-tube. If free hydrochloric acid is absent, a sufficient quantity of acid is added to cause the appearance of the Congo reaction. This mixture is poured on discs of albumin about 1.5 mm. thick and 10 mm. in diameter. These discs can be cut from hard-boiled egg with a suitable knife or a cork-borer or with a goose-quill that is cut off straight. The test-tube is placed in the incubator at blood-temperature, and it is determined how much time is required to dissolve these discs of albumin. If sufficient pepsin is present, these discs are usually dissolved in from one-half to one hour; at the expiration of this time the solution becomes clear and contains no flakes of albumin; this test can be considered valid only if the discs of albumin are completely dissolved.

Instead of using discs of albumin,[1] washed raw fibrin or fibrin colored with carmin, according to Grützner, may be employed. I believe, however, that the discs of albumin are more appropriate for this purpose, for the reason that they are more uniform in consistence than fibrin. If the discs of albumin are always of a uniform size, the time required to dissolve them fluctuates within very narrow boundaries, provided the stomach-contents is normal.

As a rule, it is unnecessary to perform quantitative estimations of pepsin. The methods employed for this purpose are those of Leube, Grützner, Brücke,[2] Jaworski,[3] Hammerschlag,[4] and others. Leube's method is a simple one, but the results obtained are rather indefinite. It does teach us something, however, in regard to the effects of the administration for therapeutic purposes. Leube's method is the following: To two equal portions of stomach-contents the same quantity of albumin is added. To one of the two fluids a small quantity of pepsin is added; if the fluid containing the additional amount of pepsin digests the albumin more rapidly than the other, this is considered a proof that pepsin is deficient in the stomach-contents.

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[1] Fibrin or discs of albumin may be preserved in glycerin; before using them they should, however, be carefully freed from any glycerin that may cling to them by repeated washing in distilled water in a test-tube. It is permissible to preserve the discs of albumin in distilled water for a few days at least, provided the surrounding temperature is not too high.

[2] Brücke, *Sitzungsberichte der Wiener Akademie*, vol. xxxvii., p. 131.

[3] Jaworski, *Münch. med. Wochenschr.*, 1887, No. 33.

[4] Hammerschlag, *Versamml. deutscher Naturforscher und Aerzte*, Vienna, 1894; *Internat. klin. Rundschau*, 1894, vol. viii., No. 39; *Wien. klin. Rundschau*, 1895, No. 23.

Jaworski's[1] method is a useful one when the secretion of hydrochloric acid is deficient. Two hundred c.c. of $\frac{1}{10}$ normal hydrochloric acid are poured into the empty stomach in the morning. After half an hour this fluid is pumped out. The filtrate of the stomach-contents is standardized for $\frac{1}{20}$ normal hydrochloric acid by the addition of official hydrochloric acid, and diluted in a series of flasks with $\frac{1}{20}$ normal hydrochloric acid until it no longer digests, in twenty-four hours, a disc of albumin weighing 1 to $1\frac{1}{2}$ cg., 10 c.c. of the digestive fluid being employed for each test. Dilute hydrochloric acid solutions, as Jaworski has shown, possess the property of extracting pepsin, or, better, propepsin, from the gastric glands.

The method of Hammerschlag, which has recently been published, is probably better than the above for the quantitative determination of pepsin. This method is carried out as follows: First, two samples of 10 c.c. each of a 1 per cent. albumin solution containing 4 pro mille of free hydrochloric acid are poured into separate vessels. To one is added 5 c.c. of water; to the other, 5 c.c. of stomach-contents. Both flasks are allowed to remain in the incubator for one hour, and at the expiration of this time the albumin is determined in both by Esbach's albuminometer. The liquid to which water was added indicates the original amount of albumin present, and the difference between the two indicates the amount of albumin that has been digested. This difference can be expressed in percentage figures.

This method enables us readily to determine the reduction in the peptonization powers of the stomach. For ordinary practical purposes the simple digestive test described above (if necessary, after the addition of hydrochloric acid in cases of absence of free hydrochloric acid) is sufficient.

[The method of Mett is now being used more widely than any other quantitative method. It consists in sucking fresh egg-albumen into capillary tubes 1 or 2 mm. in diameter, coagulating the albumin by boiling, then cutting off portions, 3 to 5 cm. long, of the filled tubes, and adding these pieces of the tubes to gastric contents. The tube containing this is then kept at body-temperature in the incubator for ten hours. At the end of this time each end of the tubes will show a lack of solid albumin, owing to its digestion, while in the central portion some will remain. Both the empty portions and the portion still full are carefully measured, and the activity of peptic digestion determined thereby. Borisson states that the relative amount of actual pepsin present varies according to the square of the length of the empty portion of the tube, the figures for the latter being expressed in millimeters—*i. e.*, 3 mm. digestion equals 9 parts pepsin; 2 mm. equals 4 parts pepsin.—Ed.]

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[1] Jaworski, *Verhandl. d. VII. Cong. f. innere Med.*, Wiesbaden, 1888; *Wien. med. Presse*, 1888, Nos. 48, 49.

Rennet and Rennet Zymogen.—Normal gastric juice contains rennet and rennet zymogen in addition to pepsin and hydrochloric acid. The determination of rennet and of its precursor, rennet zymogen, is of no diagnostic significance. Rennet, as we know, possesses the power of curdling milk independently of hydrochloric acid. This property is utilized for determining the presence of rennet. It is necessary, in order to get valid results, not to allow the fluid to become too acid. According to Leo,[1] the determination can be carried out as follows: Three or four drops of the stomach-contents filtrate are added to 5 to 10 c.c. of milk, and the whole allowed to stand in the incubator for from ten to fifteen minutes. A more accurate method is to neutralize 5 to 10 c.c. of stomach-contents with $\frac{1}{10}$ normal sodium hydrate solution, and to mix with 10 c.c. of neutral or amphoteric raw, or better boiled, milk. If the mass forms a flocculent coagulate after standing for from one-quarter to one-half hour in the incubator, the presence of rennet is revealed.

In order to demonstrate the presence of rennet zymogen, the ferment itself is destroyed by alkalinizing 10 c.c. of stomach-contents with $\frac{1}{10}$ normal sodium hydrate solution; 10 c.c. of milk and from 3 to 5 c.c. of a 1 to 2 per cent. solution of chlorid of calcium are then added, and the mixture placed in the incubator. If the zymogen is present, casein is precipitated after a few minutes. We believe that more than from ten to twenty minutes are required for this, although most authors state that the reaction should occur within this time. Dr. Johnson[2] has performed some experiments in my laboratory which show that coagulation may not occur for a longer time, particularly if boiled milk is used instead of unboiled milk. Boas has devised a method of quantitatively determining rennet by estimating the degree to which the stomach-contents can be diluted before it loses its power of curdling milk. Boas,[3] Johnson, and Klemperer[4] have done much work on the clinical significance of rennet. Boas found that in a healthy subject the stomach-contents can be diluted in the proportion of 1 : 100–150 c.c., but that in cases of secretory insufficiency, a dilution of 1 : 5 or 1 : 10 prevents the action of rennet. Boas interprets the prognostic value to these quantitative determinations of rennet in the following sense: If rennet becomes inactive when stomach-contents is diluted, as 1 : 5 or 1 : 10, he claims that the chances for recovery are not so good as if a dilution of 1 : 50 can be tolerated.

[Mennier[5] has found that rennet disappears entirely in case of gastric carcinoma, and while it is much diminished in a simple gastritis of equal degree, it is not entirely absent. He feels that the prognosis is influenced according to the amount of rennet present in the gastric juice. This conclusion seems to be substantiated by the test of clinical experience, and while it cannot be called a conclusive test, it can be regarded as evidence of some value.—Ed.]

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[1] *Berlin. klin. Wochenschr.*, 1888, No. 49.

[2] Johnson, "Studien über das Vorkommen des Labferments," *Zeitschr. f. klin. Med.*, vol. xiv.

[3] Boas, "Labferment und Labzymogen im gesunden und kranken Magen," *ibid.*

[4] Klemperer, "Die diagnostische Verwerthbarkeit des Labferments," *ibid.*

[5] *Boas' Arch.*, vol. vii., No. 3.

The Examination of Proteid Digestion.—The determination of the different stages of hydration of albumin is so far of no practical value. In the first place, the methods for determining the different intermediary stages in the digestion of albumin are too complicated for practical purposes; on the other hand, an exact knowledge of these stages is of no value in diagnosis. The value of the biuret reaction is certainly overestimated. This is a color-test; if an excess of KOH and a few drops of a copper sulphate solution are added to stomach-contents, a peculiar, rosy-reddish color will appear. This demonstrates that peptic digestion has occurred, but does not give us any information in regard to the degree of the digestion of albumin. Any stomach-contents will give a more or less pronounced biuret reaction, but we cannot draw any conclusions in regard to the strength of the gastric juice from its appearance.

The Examination of the Digestion of Starch.—The determination of the starch digestion is quite important—certainly more important than the determination of the degree of albumin digestion. It is true that the digestion of starch is not a function of the stomach, but of the salivary glands; the ferment of these glands at first converts starch into soluble starch, then into erythrodextrin, achroödextrin, and finally maltose. At the same time we frequently find that certain stomach-diseases exercise an influence on the digestion of starch.

The salivary ferment is very sensitive to the action of acids. It first acts upon starch in the mouth, and continues its action in the stomach during the first stage of digestion—namely, the amylolytic stage—as long as the amount of hydrochloric acid is not too great. As soon as 0.12 per cent. of hydrochloric acid is present, the diastatic action of saliva stops. This applies to hydrochloric acid in any form or combination—not only to free hydrochloric acid.

If the secretion of hydrochloric acid is abnormally great or continuous, the digestion of starch in the stomach is soon stopped, and in cases of this kind we find that there is either no digestion of starch or that the end-products of saccharification have not been formed. We will usually find several of the intermediary products. The reverse is the case if there is a deficiency in hydrochloric acid—as, for instance, in the initial stages of digestion or in cases of subacidity. In testing the digestion of starch it is necessary, therefore, to determine whether the end-products of starch-digestion have been formed or whether the process was arrested after the first intermediary products had been generated.

The stomach-contents is examined for dextrose by Trommer's or Nylander's test, preferably after removing all the albumin from the filtrate. In order to determine the presence of the intermediary stages of carbohydrate digestion, a dilute iodin-potassium iodid solution is employed (so-called Lugol's solution, composed of 0.1 of iodin, 0.2 of iodid of potash, 200 distilled water). If a certain quantity of this reagent is added to filtered stomach-contents, a bluish-violet coloration of the fluid will reveal the presence of soluble starch amidulin; if it turns purple, of erythrodextrin. If achroödextrin, dextrose, and maltose alone are present, there is no change of color. If soluble starch is present, a blackish-blue precipitate is formed. As achroödextrin has a greater affinity for iodin than the other intermediary products of saccharification of starch and starch itself, the degree of amylolysis can be determined only if an excess of iodin solution is added; if an excess is not added, some of the intermediary products of dextrinization that require large quantities of iodin in order to produce the color change may be overlooked.

In cases, therefore, where carbohydrate digestion in the mouth and the stomach is normal, no color appears on the addition of Lugol's solution to stomach-contents. If a violet or a blue color appears, this shows that saccharification is deficient. Subsequent examination will have to decide whether this is due to a lack of salivary ferment or to overabundant secretion of hydrochloric acid, or to some other undetermined cause. In general, it is due to excessive secretion of hydrochloric acid. Deficient ferment action is rare, if it occurs at all.

The Digestion of Fat in the Stomach.—Marcet[1] has made it very probable that the fats are also changed in the stomach. This author claims to have observed a splitting of fats into glycerin and fatty acids. Later, Cash[2] and Ogata,[3] in Ludwig's laboratory, demonstrated that the surviving stomach and the stomach of living animals are capable of causing a slight disintegration of neutral fats. Klemperer and Scheurlen,[4] in Leyden's clinic, repeated Ogata's experiments in human subjects, and found that from 1 to 2 per cent. of oil is normally decomposed within two hours; and if the oil is allowed to remain a longer time, they found that, particularly in dilatation of the stomach with fermentation, as much as 6 per cent. of oleic acid could be formed. These experiments from Leyden's clinic are not numerous (6), so that further investigations in this regard are to be desired. The power of the stomach to split fat is quantitatively very slight.

[Volhard, working in Riegel's clinic, has recently reported a series of experiments which seem to demonstrate conclusively that there is a fat-splitting ferment secreted by the stomach. This ferment, however, has little or no action upon fats that are not well emulsified, its action being best studied with egg-yolk or milk. It is probably of little importance normally, as it is destroyed by any marked degree of acidity of the medium in which it is found, and, as stated, it acts only on well-emulsified fats, while in the stomach most of the fat of the food is not found well emulsified. Volhard believes, however, that this ferment probably causes much of the fat-splitting that occurs in complete pancreatic occlusion, a phenomenon previously attributed to the action of bacteria. The ferment was found both in gastric secretion and in extracts by the mucosa. It is apparently secreted chiefly by the mucosa of the fundus.—Ed.]

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[1] Marcet, *The Medical Times and Gazette*, new series, 1858, vol. xvii., p. 210.

[2] Cash, *Du Bois' Arch.*, 1880, p. 323.

[3] Ogata, *ibid.*, 1881, p. 515.

[4] Klemperer and Scheurlen, *Zeitschr. f. klin. Med.*, vol. xv., p. 370.

THE DETERMINATION OF THE MOTOR POWER OF THE STOMACH.

In examining the perversion of gastric function it is not sufficient simply to examine the secretory function of the stomach, to establish the nature of the perversions of gastric secretion, to obtain the history of the case and a picture of the general and the local manifestations of the disease—more is required. The general rule can be formulated that normally the secretory, motor, and the absorbing power of the stomach run parallel. In pathologic cases, however, we frequently see that the disturbances of these different functions do not run parallel; in fact, the impression is often created that the perversion of one function produces an increased manifestation of some other one. This process resembles the compensation seen, for instance, in early stages of carcinoma. Even in cases where no stenosis of either orifice of the stomach is present, we find the motility of the stomach normal or increased, although the secretory powers of the organ may be very much reduced. If the stomach is siphoned out three to four hours after a test-meal, no remnants of food will be found in the stomach. This is pathologically interesting and also of practical significance. It seems that the stomach, if its peptic power is reduced, attempts to compensate this reduced power by developing correspondingly greater energy in expelling its contents. This causes the food to enter the intestine before it is decomposed, and von Noorden[1] has shown that the intestine can vicariously assume the peptic functions of the stomach. An example of the opposite condition is seen in certain forms of hypersecretion combined with ectasy. Although here we find an increased secretion of gastric juice, the expulsion of the stomach-contents is slow. In other cases again secretory and motor functions are altered in the same sense —for instance, in ulcer, in which increased secretion of hydrochloric acid and increased motility run parallel.

These examples show that it is not sufficient to test the secretory powers of the stomach alone, and that an understanding of these powers is not enough to allow us to draw any conclusions in regard to the powers of motility. In every case of stomach-disease both secretory and motor powers should be examined.

Impairment of the motor powers is as significant as impairment of the secretory powers—in fact, may be more important. If the motor power is intact, a deficiency in the secretory powers of the stomach may be compensated by intestinal digestion; on the other hand, a deficiency in the motor powers cannot be compensated, and any severe degree of disturbance in this direction will impair general nutrition.

Before describing the methods for testing the motor powers of the stomach I will briefly describe a few recent contributions to the physiology of this function. A knowledge of these facts will enable us to understand certain pathologic processes that we must discuss later on.

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[1] *Zeitschr. f. klin. Med.*, vol. xvii.

The motor function of the stomach is two-fold—namely: (1) thoroughly to mix the food with the stomach secretions, and (2) to propel it onward into the intestine. The respiratory movements aid this function, and other movements that are transferred to the stomach from other organs assist in the same manner. There is a great diversity of opinion in regard to the active movements of the stomach proper. There can be no doubt, however, that there is a regular, wave-like motion, particularly in the region of the pylorus. It is questionable whether the observations of Hofmeister and Schütz[1] on the stomach of dogs that had been recently killed can be compared to the conditions normally existing in human beings during life. According to these experiments, two phases in the movements of the stomach can be distinguished: the first begins at the fundus, and consists in the universal contraction of the circular musculature of this part; at the same time vertical contractions proceed from the cardia and the pylorus, which are strongest at the greater curvature and near the pyloric part of the stomach. A second phase is seen exclusively in the region of the pylorus; here the longitudinal and the circular musculature contract.

However interesting these experiments may be, they give us little information in regard to the effect of these contractions on the expulsion of stomach-contents. Hirsch[2] has attempted to answer this question by certain animal experiments. According to clinical observation, we are justified in assuming that the stomach-contents is emptied in stages. Rossbach's[3] experiments on dogs, however, show that movement begins in a full stomach at an early stage of digestion, but that some four to eight hours after the ingestion of the meal the pylorus is opened and then the stomach-contents is rapidly expelled in several successive movements. More recent experiments by Hirsch seem to point in another direction. This investigator saw that the stomach of dogs emptied itself in stages, that the propulsive movements occur at long and short intervals (one-quarter of a minute to several minutes), and that these movements begin a short or a long time after the ingestion of food, depending altogether on the character of the material eaten. The expulsion of chyme, according to Hirsch, is altogether dependent upon the rapidity with which the food is liquefied in the stomach.

The statements in regard to the influence of gastric acidity on the motor functions of the organ are contradictory. Brücke formulated the theory that the acid of the stomach causes the movements of the organ, and to this day many authors believe in this view. An argument against this theory is the fact that peristalsis begins very soon after the ingestion of food, and at a time when no *free* acid, at all events, can possibly be present in the stomach. Von Pfungen states, in contradiction to Brücke, that alkalis cause opening, and acids closure, of the pylorus. Hirsch[1] has recently published some exhaustive experiments on these questions, showing that both neutral and alkaline watery solutions were propelled into the duodenum from the stomach within a short time after their ingestion. Hirsch concludes from these experiments that neither alkaline, neutral, nor acid fluids can exercise any influence on the opening or closing of the pylorus. He believes that this process is altogether independent of the reaction of the stomach-contents.

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[1] *Arch. f. exp. Pathol.*, vol. xx.

[2] *Centralbl. f. klin. Med.*, 1892, No. 47.

[3] *Deutsch. Arch. f. klin. Med.*, vol. xlvi.

I am of the opinion that all these experiments cannot be applied to man. All experience certainly indicates that acidity exercises a certain influence on the motor functions of the organ. We need only refer to spastic contractions of the pylorus, seen in cases of increased secretion of hydrochloric acid.

Recently von Mering[2] has published some experiments that are interesting, particularly as they correspond very well with clinical observations.

Von Mering corroborated the discoveries of Hirsch and others that the stomach-contents is propelled into the intestine in intervals—namely, by rhythmic opening and closing of the pylorus; he, therefore, refutes the views of Rossbach. Fluids leave the stomach more rapidly than solid food; an empty stomach absorbs no water; alcohol is absorbed in large quantities, watery solutions of sugar in moderate quantities, alcoholic solutions of sugar in larger quantities. Dextrin and peptone are absorbed in the stomach. The amount of a substance absorbed is proportionate to the concentration of its solution.

The absorption of the substances enumerated is accompanied by an active secretion of water into the stomach that is, as a rule, proportionate to the amount of substance absorbed. Even though no hydrochloric acid is found in the stomach, there may be an abundant secretion of water.

These results promise to be of great practical value. They seem to explain the well-known fact that cases with pronounced gastrectasy as a result of stenosis of the pylorus frequently suffer from thirst, oliguria, constipation, and dryness of the skin. Formerly this was explained by the assumption that a dilated stomach absorbed water with difficulty and that little water entered the intestine through the narrow pylorus. Von Mering's experiments, however, show that very little water is absorbed in the stomach, but that certain food-products are absorbed, and that water is secreted to take their place. In cases of stenosis of the pylorus, therefore, more fluid may be found in the stomach than was taken. The dilatation of the stomach seen in stenosis of the pylorus is, therefore, caused by the stagnation of the ingesta and by the increase of the stomach-contents following the pouring-out of water into the stomach from the blood.

Moritz[3] corroborated the results of all those experiments. He experimented on dogs, and, like von Mering, found that water brought into the stomach was immediately expelled in jerks. On an average, three contractions of the stomach occur every minute. The contractions are rhythmic, and are separated by regular intervals; they persist uninterruptedly until the stomach is empty. On introducing fluids and different solid substances and mixtures of the two, the following results were seen: The slight movements of the stomach bring about a separation of the stomach-contents by propelling the softer portions toward the pylorus and leaving the more solid ones behind. The sudden respiratory changes of pressure seem to aid in mixing the stomach-contents. From the pyloric portion of the stomach the contents of the organ is suddenly and forcibly pushed into the duodenum in movements that are separated by regular intervals. Fluids, particularly water, are rapidly and almost completely evacuated. Later, mushy portions are poured out; the solid portions are retained until they too become mushy and are then forced out into the duodenum.

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[1] *Centralbl. f. klin. Med.*, 1893, Nos. 4, 18.

[2] *Therapeut. Monatsh.*, 1893, and *Verhandl. d. XII. Cong. f. innere Med.*, Wiesbaden, 1893.

[3] *Münch. med. Wochenschr.*, 1893, No. 38; *Berichte d. 65. Versamml. deutscher Naturforscher und Aerzte*, Nuremburg, 1893.

This teaches us that in cases of impaired gastric function food should be given in a form that is readily movable by the stomach, for the digestibility of a given article of food is dependent on the length of time it remains in the stomach.

Another interesting question had to be solved, namely, whether the physical consistence of the stomach-contents alone determined its propulsion into the duodenum, or whether certain chemical properties of the ingesta played a rôle. Moritz's[1] experiments on human subjects decided the question in the latter sense. In my own experiments water was seen to leave the stomach in a surprisingly short time. In the case of other fluids, particularly if they contain much carbonic acid gas, if they were very alkaline or very acid, they remained a much longer time within the organ; the same applied to bouillon, beer, milk, and oil. It was also shown that water was evacuated at a much later period if solid substances (rolls, meat) were administered at the same time. The irritation caused by the solid admixture produced, on the one hand, an increased secretion of gastric juice, and, on the other hand, a retardation of the propulsion of stomach-contents.

It would lead us too far to enter further into the physiologic questions involved. We wish merely to enumerate a few facts that are pathologically significant. We possess a number of methods for testing the motor function of the stomach.

Method of Leube.—The oldest method is that of Leube.[2] This author administers a test-meal consisting of a plate of soup, a beefsteak, and a roll. If the stomach was found empty after seven hours and if nothing could be washed out by lavage, Leube assumed that the time of digestion was normal. Nowadays we express ourselves differently and better, by saying that the motor power is normal. Leube has determined that a healthy stomach is empty seven hours after the ingestion of such a test-meal. According to our views to-day, the fact that the stomach is empty at this period does not necessarily indicate that the organ is normal; it does indicate, however, that its motor power is sufficiently active. The motor power may, however, be good, and the secretory power at the same time be very much impaired.

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[1] *66. Versamml. deutscher Naturforscher und Aerzte*, Vienna, 1894; abstract in *Münch. med. Wochenschr.*, 1894, No. 41.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii.

This method is undoubtedly the most natural, the most reliable, and, at the same time, the most simple, one for determining the motor power of the stomach. If the food remains in the stomach for a longer period of time, this indicates that the motor power is reduced. At the same time we cannot say that the motor power is absolutely reduced, for the impairment may be relative. In cases of stenosis of the pylorus, for instance, with secondary ectasy, the food remains in the stomach for a long time, although the violent peristaltic movements that we frequently see through the abdominal walls indicate that the motor power of the stomach is, if anything, exaggerated. Postmortem examination will in many cases reveal considerable hypertrophy of the gastric muscularis, chiefly in the pyloric part of the organ.

If the food remains in the stomach for an abnormally long period of time, this demonstrates that there is an insufficiency in the sense that the motor power of the organ is not sufficient to expel the contents within the normal time. This insufficiency need not, however, be an absolute one; it may be relative, and is, in fact, frequently relative. It is important from a practical point of view to distinguish between absolute and relative insufficiency of this kind, and the treatment of the motor insufficiency will be different according to which form is present.

The general rule may be formulated that the motor power of the stomach should be tested by siphoning out the stomach-contents seven hours after a test-meal. The practitioner will, however, generally examine both the secretory and motor power at the same time. We have seen that it is impossible to make the necessary tests for the secretory power of the stomach by removing the test-meal at any given specified time after the ingestion of the meal. In cases of ectasy of high degree a different time will be selected than in cases of simple hyperacidity. If the physician is experienced, it will usually be an easy matter to select the correct time for removing the stomach-contents for diagnostic purposes. According to the character of the disease and the general symptom-complex, the stomach-contents will be removed after three or four, sometimes five or even six or seven, hours. As a rule, it is possible to formulate a judgment in regard to the motor power of the stomach from the quantity of gastric contents obtained for chemical examination. If, for instance, a teaspoonful of a thin, pultaceous mass is pumped out four or five hours after a test-meal, the motor power can be considered good; if, on the other hand, large quantities of stomach-contents are found six hours after a test-meal, we will know that the motor power is either absolutely or relatively decreased.

In many cases it is desirable to determine the exact degree of this impairment of motor power or atony. For this purpose Boas has described a special test-supper. I have employed a similar meal for several years, long before Boas recommended his test-supper. If it is found that seven hours after a test-meal much food is still left in the stomach, the motor power is reduced. We do not know, however, whether the stomach could or could not get rid of a smaller meal within the same time or a longer time. As a matter of fact, it will be seen that in some cases a simple supper taken after the stomach has been thoroughly washed out is expelled by the stomach during the night, so that the stomach is found empty the next morning. In other cases again a considerable quantity of food will be found under the same conditions. Both cases, therefore, react in the same manner to a test-meal in the sense that an abundant quantity of substance is found seven hours afterward. In the case of the test-supper, however, there were certain differences, as the stomach of one group was found empty in the morning, whereas in the other group a considerable portion of food remained in the stomach ; in other words, the latter group was enabled to get rid of the small meal during the night. Both cases are atonic, and in both the motor power is reduced ; the former case, however, has a mild degree of this affliction ; the latter, the highest degree of atony. I have called attention to the necessity of distinguishing between these two degrees of atony for many years, and have called attention to the significance of this test in general practice. The test-supper may be different. Boas recommends cold meat with rolls and butter and a large cup of tea.

It is self-evident that some other combination of food can be used for determining the motor power, just as we have a certain latitude in selecting the composition of a test-meal. An ordinary test-breakfast may be employed. Normally this should be out of the stomach within two hours.

I have a particular reason for preferring the test-meal. The test-breakfast does not tax the secretory power of the stomach very much ; the same applies to the motor power. Only in cases of great insufficiency of the gastric musculature will we find considerable quantities of a test-breakfast two or three hours after its ingestion. Cases that are as severe as this are easily diagnosed ; but in practice we wish to diagnose not only the severe cases, but also mild cases of atony, and for the diagnosis of the latter condition the test-meal is decidedly better.

Although the method described for testing the motor power of the stomach is the most simple and the most natural one, other methods have been sought. I mention them here merely for completeness' sake. They would be preferable only if they were easier to execute or if they gave better results. As a matter of fact, they do not excel in either respect, and are in no way superior to the above-mentioned method of determining the motor power of the stomach from the time required for the digestion of a stated quantity of food.

Klemperer's Oil Method.[1]—This method is based on our knowledge that liquid fats are not absorbed by the stomach. The method is executed as follows: 100 gm. of olive oil are poured into the stomach after the organ has been thoroughly washed out. Two hours afterward the stomach-contents is aspirated and the organ washed out with water. The oil is separated from the water in a separating funnel, and is measured. The deficit indicates the amount of oil that has passed into the duodenum. In normal subjects Klemperer found that from 70 to 90 gm. normally pass into the intestine within this time; if the motor power was reduced, smaller values were found. Klemperer himself does not advocate this method for general practice; in fact, it is hardly suitable for practical purposes, nor is it a simple method. Finally, certain objections can be formulated against it.

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[1] *Deutsch. med. Wochenschr.*, 1888, No. 47.

The same applies to the method of Mathieu and Hallot,[1] which has recently been published. The method is as follows: 10 gm. of an emulsion of oil are administered to the patient, together with a test-breakfast. The quantity of fluid present in the stomach is determined according to the method of Mathieu-Remont. The stomach-contents is then expressed, the oil extracted with ether, and weighed. It can now be readily determined how much oil has disappeared, and how much fluid has left the stomach. From the quantity of oil that is recovered the existence or non-existence of a condition of stasis is determined.

The Salol Method of Ewald and Sievers.—Ewald and Sievers[2] recommend salol for testing the motor power of the stomach. As this substance is indifferent to acid solutions, but is decomposed to salicylic acid and phenol in weak alkaline solutions, it is not decomposed in the stomach, but is split as soon as it comes in contact with the alkaline fluids of the intestine. This splitting is manifested by the appearance of salicyluric acid in the urine. The presence of this substance in the urine can be determined by the addition of a little neutral chlorid of iron solution, which stains the urine violet. If 1 gm. of salol, preferably in a capsule, is given to a healthy subject, the reaction appears in the urine some time between half an hour to seventy-five minutes after the ingestion of the drug. If the reaction appears later than this, we know that the salol did not enter the intestine within a normal time.

Brunner[3] repeated Ewald's experiments shortly after the publication of this author appeared, and found that the results were not uniform. At that time I formulated a number of objections against the general applicability of salol for determining the motor powers of the stomach. Other authors indorsed these objections (Huber,[4] Decker,[5] Wotitzky,[6] Reale and Grande,[7] Stein,[8] and others). Stein, above all, demonstrated that salol can be absorbed from the stomach if the organ is closed, even if the reaction is acid, and that under these conditions the decomposition products of salol appear in the urine. He also showed that an increased secretion of mucus causes a decomposition of salol. Huber, therefore, attempted to use the salol test in a different way, by determining the motor power of the stomach not from the delay in the appearance of decomposition products of salol, but from the length of time during which salol reactions persisted in the urine. He found that in a healthy subject the reaction could be elicited after twenty-six or twenty-seven hours, but that in patients with motor insufficiency of the stomach the reaction persisted for a much longer time (three, six, twelve, and more hours). Huber's modified method is a very simple one. One gm. of salol in capsule is administered with the midday meal. On the following day, about twenty-seven hours later, the bladder is emptied. If salicylic reactions are found three, six, or more hours after this period, this indicates motor insufficiency. Huber was enabled to determine that in 2 cases of more or less severe motor insufficiency reactions for salol persisted for an abnormal time.

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[1] Mathieu and E. Hallot, "Note préliminaire sur un moyen clinique de mesurer la motricité gastrique et le transit des liquides dans l'estomac," *Cong. d. méd. interne*, Lyons. Quoted from Boas, *Arch. d. Verdauungskrankheiten*, vol. i., No. 2.

[2] *Therapeut. Monatsh.*, August, 1887.

[3] *Deutsch. med. Wochenschr.*, 1889.

[4] *Correspondenzbl. f. schweizer. Aerzte*, 1890.

[5] *Berlin. klin. Wochenschr.*, 1889.

[6] *Prager Wochenschr.*, 1891.

[7] *Rivista clinica*, 1891; *Wien. med. Wochenschr.*, 1893.

[8] *Ibid.*, 1892.

This modification of the salol method is not universally recognized, and even if it were free from all objections, could hardly replace the older method of Leube.

Dehio's method[1] has been employed for testing the motor power of the stomach, although it was originally devised for determining the topography of the organ. If a patient with atony of the stomach drinks two or three glasses of water in succession, and if the extent of the stomach dulness in the erect position is determined after each glass, it will be found, as a rule, that the progressive filling of the stomach with water causes the area of dulness to descend lower than normal. The method of Rosenbach,[2] which we have described above, for determining the position of the stomach, has also been employed for testing the mechanical powers of the organ. Both methods, it is true, indicate the amount of resistance that the stomach can offer to distention, but they certainly give us no positive and reliable information in regard to the motor powers of the organ.

[**Iodipin** has recently come into use as a test for gastric motility, based on the investigations of Winckler and Stein.[3] Iodipin is not decomposed by the gastric juice, but is by the action of bile and pancreatic juice. Therefore, according to the rapidity with which free iodin is detected in the urine or saliva, one may draw conclusions concerning the gastric motility. Normally, the iodin can be detected in the saliva fifteen minutes after ingestion, whereas in diminished motility it may not appear for some hours after. When free iodin is not found in the saliva within forty-five minutes after the iodipin is swallowed, it may be concluded that the motor function of the stomach is delayed. Heichelheim[4] found that in hyperacidity the reaction rarely occurred before three-quarters of an hour, while in achylia it occurred quite early;

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[1] *Verhandl. d. VII. Cong. f. innere Med.*, 1888.

[2] *Volkmann's Samml. klin. Vorträge*, 1878, No. 153.

[3] *Centralbl. f. innere Med.*, No. 33.

[4] *Boas' Arch.*, vol. vii., No. 3.

and in 3 cases of icterus the test did not work even after four and a half hours, owing, it is supposed, to the absence of bile in the duodenum. Heichelheim regards the method as the most reliable next to Riegel's method.—ED.]

I refer to the section on Atony and Ectasy of the Stomach for the causes of motor insufficiency. In this place we will only call attention to the fact that two forms of motor insufficiency are distinguished in practice: in one there is a real loss of muscular power; in the other there is no loss of muscular power, but there is some abnormal obstacle to the propulsion of chyme.

In the latter case muscular power may even be increased. Notwithstanding, this complete compensation may not be present and the motor power of the stomach may be insufficient. As a rule, this condition is found in stenosis of the pylorus or of its vicinity. The latter case, therefore, is a *relative* motor insufficiency; the former one, *absolute* insufficiency.

The Gastrograph of Einhorn.—I may briefly mention in this place an apparatus that Einhorn[1] has recently described to register the movements of the stomach. It does not register the propulsive movements of the organ that are intended to move the food into the intestine as much as the smaller movements that mix and break up the stomach-contents. Einhorn's apparatus is capable of registering the most minute movements of the latter character. He calls it a gastrokinesograph or a gastrograph. The practical value of this method of examination has so far not been demonstrated. Observations made with this apparatus show that in pathologic cases the motor power of the stomach is sometimes reduced, sometimes increased. Einhorn himself has so far not reported any exhaustive details of this method of examination; it may suffice, therefore, merely to mention the apparatus in this place.

METHOD FOR TESTING THE ABSORPTIVE POWERS OF THE STOMACH.

The stomach, in addition to its secretory and motor powers, possesses absorptive powers; one of its functions is to absorb certain portions of its contents and to pour them into the blood and the lymph. Whereas we are in the habit of testing the motor and the secretory powers of the organ in every case of serious disease of the stomach, provided, of course, that the passage of the stomach-tube is not contraindicated, we do not commonly test the absorptive powers. This is due to the fact that our knowledge of the normal absorptive powers of the stomach is still so insufficient. It is true that the splendid experiments of von Mering[2] demonstrate that an abundant quantity of alcohol, a moderate amount of sugar and dextrin, peptone, albumose, etc., can be absorbed, and we know, further, that the amount of these substances absorbed is more or less dependent on the concentration of their solutions (Brandt[3]). From all this we can naturally assume

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[1] *New York Med. Jour.*, September, 1894; and *Zeitschr. f. klin. Med.*, vol. xxvii.

[2] *Therapeut. Monatsh.*, 1893.

[3] *Zeitschr. f. Biol.*, 1893, vol. xxix.

a priori that in certain pathologic conditions, as in catarrh or inflammation, the absorptive powers of the organ will be impaired. No decisive investigations of this subject have, however, so far been published. A certain insight into the degree of absorptive power may be obtained if the stomach-contents is quantitatively examined for digestive products, peptone, sugar, etc., which are still present after a certain time has elapsed.

Heretofore the absorptive powers of the stomach for potassium iodid alone have been tested for clinical purposes. The most common method is that of Penzoldt[1] and Faber.[2] It is executed as follows: 0.2 of potassium iodid is administered in a gelatin capsule on an empty stomach; the saliva and the urine are examined every few minutes for iodin with starch paper and fuming nitric acid. As soon as the first trace of iodin appears in the urine, a distinct blue color appears on the paper.

Normally, from six and one-half to fifteen minutes elapse between the ingestion of the iodid and the appearance of the first iodin reactions in the urine. In pathologic cases the appearance of this reaction may be very much retarded.

Our[3] own experiments show that in a healthy subject the average time of absorption for iodin is thirteen and a half minutes. We examined the urine more frequently than the saliva, and demonstrated the presence of iodin by adding fuming nitric acid and afterward shaking with carbon disulphid—this causes a reddish-violet color to appear. I was enabled to determine, however, that different results were obtained if the iodid of potash was given on an empty stomach at the beginning or at the height of digestion. I found that absorption was quickened if the drug was given in the later stages of digestion and on an empty stomach than if it was given immediately after the ingestion of a meal. Malinin[4] also found that the time of absorption varied if the drug was taken on an empty stomach or a full stomach. When the stomach was empty, absorption was quicker than if the stomach was full. In pathologic cases I have frequently found, although not always, that the time of absorption was longer, particularly in ulcer of the stomach.

This test with iodin is hardly a reliable one in making a differential diagnosis, nor will it ever be universally employed as an indicator of the absorptive powers of the stomach. As a matter of fact, the method described is not a popular one, however interesting the knowledge obtained in regard to the absorptive powers of the stomach may be. In practice, we are more interested in determining to what extent the disease of the stomach interferes with digestion and assimilation. The iodin test gives us no information in this respect. It is self-evident that the gastric digestion of proteids, and consequently the absorption of these bodies, is interfered with in cases of subacidity and of anacidity. Changes in the chemism of the stomach, on the other hand, are not so important for the digestion of the fats and carbohydrates. The experiments of von Noorden show that even if large quantities of fats and albumin are ingested, they never appear in excessive quantities in the stools. Such favorable conditions are, of course, seen only if the stomach has the power of propelling its contents into the intestine within a normal time.

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[1] *Berlin. klin. Wochenschr.*, 1882.

[2] Faber, *Inaug. Diss.*, Erlangen, 1882.

[3] Quetsch, *Berlin. klin. Wochenschr.*, 1884.

[4] Malinin, "Ueber den Einfluss des vollen und leeren Magens auf die Schnelligkeit der Resorption einiger Medicamente und deren Ausscheidung," *Wratsch*, 1894, No. 37.

THE EXAMINATION OF THE CONTENTS OF THE STOMACH AFTER FASTING.

It is not always sufficient to determine the conditions in regard to the quantity, the consistency, the chemical changes, of the stomach-contents at the height of digestion; occasionally it may become necessary to investigate these questions when the stomach should be empty —*i. e.*, early in the morning before the patient breakfasted. At this time a normal stomach will be found to contain only a few drops of a neutral or acid fluid; if, therefore, large quantities of material are found in the morning, this is pathologic under all circumstances. In most diseases of the stomach, however, the organ will be found empty in the morning.

Schreiber[1] has stated that the normal stomach secretes gastric juice continuously and without any irritation from ingesta or other causes. He also claims that early in the morning a little secretion will always be found. In these statements this investigator places himself in direct opposition to the general consensus of opinion and also to my own findings in normal subjects. For a more detailed discussion of this question I refer to the section on Hypersecretion. I believe that in the great majority of stomach-diseases only very little gastric secretion is found after fasting for some time. In certain diseases, on the other hand, a large or small amount of gastric juice may be discovered in the stomach even after a considerably prolonged fast.

It is important that we should know the states in which we may expect to find some secretion in the stomach in the morning before breakfast, as this knowledge may aid us greatly in making our diagnosis, and we are considering chiefly diagnostic points of view in this section. This question may be answered in general as follows: The stomach should be examined in all cases where we are justified in suspecting that an abnormal quantity of gastric contents will be found in the morning after fasting. It is difficult to answer the question for each individual case. An examination of this character should be undertaken:

1. In all cases of ectasy and atony of high degree in which it is known that after a test-meal or a test-breakfast the ingesta remained in the stomach for an abnormally long time. It is immaterial whether the disease of the stomach is due to carcinoma, cicatricial stenosis, continuous secretion of gastric juice, or any other cause. We can proceed in two ways—the stomach is either washed out in the evening and then pumped out early in the morning, or, better, the stomach is thoroughly washed out

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[1] *Deutsch. med. Wochenschr.*, 1893, Nos. 29, 30; 1894, Nos. 18, 20, 21.

before supper. After the first washing the stomach is filled with water and the patient is instructed to express the stomach-contents; or, again, the stomach may first be washed out while the patient is sitting up; later he should be instructed to lie down, and in this position the stomach thoroughly washed out (Fleiner). In many instances a considerable amount of food will be thus obtained, even though the water was clear while the patient was sitting up. After the stomach has been thoroughly cleansed in this way a simple supper is administered and the stomach-contents aspirated and examined the next morning before breakfast.

In cases of this kind the chief purpose of the morning examination is to test the motor powers and not the secretory powers of the stomach, for this question has presumably been decided by previous examinations of stomach-contents after the administration of a test-breakfast or a test-meal. We can diagnose motor insufficiency of high degree if remnants of a simple meal that was administered in the evening are still found the next morning.

2. There are other cases besides ectasy or atony in which a morning examination of the stomach-contents is indicated, particularly where hypersecretion is suspected. We are justified in considering the possibility of such a state if the material washed out in the evening separates into three layers, contains an abundant quantity of amylaceous material or meat residue, has an acid reaction, and shows pronounced gas-fermentation. In a case of this kind we proceed as follows: What we wish to obtain is the gastric juice present in the empty stomach. The organ is, therefore, washed out late in the evening—at about 10 o'clock; care should be taken that all the wash-water is removed. No food or drink is introduced, and the next morning the stomach-contents is aspirated before breakfast and examined. The exact methods of examination will be described in the section on Hypersecretion. The disease under discussion is probably that which calls for such an examination of the empty stomach more than any other condition, and the results obtained are of fundamental diagnostic importance. There are other cases, besides, however, in which this examination is desirable—for instance, cases in which there is a permanent regurgitation of bile into the stomach. I[1] was the first to describe cases of this kind. The stomach-contents is frequently found to be bile-tinged, particularly if the fluid obtained toward the end of aspiration is examined or if vomit is analyzed. This admixture of bile, however, is not important; it is significant only in those rare cases in which the secretion of the duodenum continuously regurgitates into the stomach as a result of torsion, adhesions, neoplasms, etc., of the parts.

Honigmann[2] has described a case of stenosis of the intestine from my clinic that is particularly instructive.

In this case there was complete apepsia as long as the symptoms of stenosis of the intestine were manifest, and as long as the contents of the duodenum regurgitated into the stomach. During this time the gastric juice had lost all its digestive powers, so that the patient had become horribly emaciated. As soon as the lumen of the intestine became patent, the regurgitation of intestinal contents ceased and gastric secretion became normal.

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[1] *Zeitschr. f. klin. Med.*, vol. xi.

[2] *Berlin. klin. Wochenschr.*, 1887, No. 18.

Under normal conditions the peptonized gastric chyme is digested in the small intestine by the bile and the pancreatic juice; on the other hand, if the bile and pancreatic juice enter the stomach, all digestive processes are arrested. If the quantity of bile that enters the stomach is small, this does not occur, but if all the bile that is secreted and all the pancreatic juice enter the stomach, as in the case that we have observed following a stenosis of the intestine, gastric digestion stops; the bile precipitates the pepsin in the gastric juice and also combines with the proteid substances in the stomach, forming combinations that are very resistant to peptic digestion; in addition the pancreatic juice alkalinizes the hydrochloric acid, and in this manner renders pepsin inactive. At all events the presence of more or less bile in the stomach-contents should be investigated and should be considered in explaining the gastric symptoms of any given case; much will depend on the time in which the bile reaches the stomach and upon the quantity of bile that enters there.

Boas[1] has shown that a mixture of bile, pancreatic juice, and probably intestinal secretion is occasionally found in an empty stomach. This mixture Boas calls duodenal juice. The fluid is a grass-green, viscid, yellowish liquid containing the bile constituents. If it is not mixed with gastric juice, it contains all the biologic properties of the pancreatic secretion. It is capable of converting starch into maltose and dextrose, of converting proteids into peptones, and of splitting fats.

If small quantities of intestinal juice are aspirated from an empty stomach, this finding probably possesses no significance; if, on the other hand, there is a constant regurgitation of intestinal juice, we must at least think of the presence of some process that constitutes an obstruction to the flow of duodenal contents.

In cases where the flow of intestinal contents is impeded, or where there is some abnormal communication between the stomach and some portion of the intestine, we frequently find intestinal contents in the stomach.

Rare Abnormal Constituents of the Stomach-contents.—We have already mentioned bile and gastric juice among the abnormal constituents that are occasionally found in stomach-contents. Small quantities of bile and of gastric juice are quite frequently found in an empty stomach, also in the vomit, particularly if vomiting occurs while the stomach is empty. Sometimes a little bile is washed out if irrigation of the stomach is continued for a long time; this is due to the fact that under these conditions the pyloric closure is not so absolute. Small admixtures of bile and of gastric juice are of no significance. If, on the other hand, there is a permanent regurgitation of bile and of duodenal contents, as in the case described above, the matter may be of serious import.

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[1] *Zeitschr. f. klin. Med.*, vol. xvii.

The admixture of bile can usually be recognized from inspection of the stomach-contents. The chemical tests are, as a rule, superfluous; if they are carried out, the well-known methods of Gmelin for bile-pigment and Pettenkofer's for bile acids should be employed.

Mucus and saliva are quite frequently found in the stomach-contents; if large quantities are present, they are easily recognized, or if macroscopic inspection fails to reveal their presence, the mucin reaction may be made. Small admixtures of mucus are of no significance; large quantities, if we can exclude the swallowing of mucus, indicate some catarrhal affection of the gastric mucosa.

The presence of blood is frequently difficult to determine. If the blood is fresh, and if it is present in large quantities, it is easily recognized; if the blood is old, decomposed, and present only in small quantities, its detection may be difficult.

We have a number of methods for determining the presence of blood in the stomach and intestinal contents. The following are the most important ones:

1. Microscopic Examination.—Here the positive detection of red blood-corpuscles alone is decisive. If the blood has been in the stomach for some time, the red blood-corpuscles are destroyed either by the digestive process or by the hydrochloric acid; red blood-corpuscles are, therefore, rarely found in the coffee-ground vomit which is so often seen. The same applies to the feces in gastric hemorrhages.

2. The Test of Van Deen.—If turpentine is added to a brown solution of guaiac in the presence of blood, a blue color appears. This test is positive even if the blood-pigment is changed. In addition to blood, certain vegetable substances, like fresh potatoes, many vegetables, and certain inorganic substances, like iron salts, etc., produce this reaction; consequently it is not a suitable one for examination of the stomach-contents.

3. Heller's Test for the Urine.—A little potassium hydrate is added to the urine, the mixture boiled, and if blood is present, the phosphates that are precipitated will be colored red. This test is not applicable to stomach-contents because other pigments may be present; the same applies to the feces.

4. Teichmann's test, according to Weber,[1] is not suitable for stomach-contents. It consists in the microchemical preparation of crystals of hemin. The reason why this test cannot be used in stomach-contents is the fact that only a very small quantity of the mass to be examined can be used for each test, and as the blood is not evenly distributed in the stomach-contents, a small quantity of material may not show the reaction, and blood still be present in the stomach.

5. The Spectroscopic Method.—According to Weber, the method of performing an analysis of the stomach-contents by spectroscopy is the following: A few drops of concentrated acetic acid are added to the material that is to be examined. It is well previously to dilute the substance with a little water in a test-tube. The mixture of acetic acid, stomach-contents, and water is then shaken with about $\frac{1}{5}$ volume of ether. The test-tube is allowed to stand for a few minutes until a clear brown layer of ether separates on the top of the fluid; if this separation is retarded or if the upper portions of the fluid remain foamy and do not become translucent, a few drops of alcohol added to the fluid will aid the process. The brownish-red color of the extract is due to the presence of hematin in solution; this body forms an acetic ether that is soluble in sulphuric ether. This solution of hematin in acetic ether shows four absorption-bands—(1) one in the red; (2) one in the yellow; (3) one on the boundary between the yellow and green; and (4) one on the boundary between the green and the blue. The absorption-band in the red is the darkest and the most distinct one; this red band may not be due to the presence of blood, but may be caused by the chlorophyll of the food. Weber, therefore, recommends dissolving the blood-pigment present in the acid ethereal extract in alcoholic potassium hydrate solution, and then making a watery alkaline solution that can be reduced with ammonium sulphid. On the addition of ammonium sulphid the fluid becomes red, and two bands in the green of the spectrum, which are characteristic for reduced hematin, appear. The spectrum of chlorophyll is not changed by this procedure.

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[1] Weber, "Ueber den Nachweis des Blutes im Magen- und Darminhalt," *Berlin. klin. Wochenschr.*, 1893, No. 19.

All the tests mentioned above with the exception of the last one are unreliable, for reasons stated, and the last method is certainly too complicated for practical purposes. A good spectroscope is necessary, of course, and but few practitioners have one. The most convenient method for detecting blood in stomach-contents is probably a modification of Van Deen's test, as recommended by Weber. We mentioned before that this test is not suitable for determining the presence of blood in stomach-contents, because other substances give the same reaction.

Weber's modification is the following: As in the spectroscopic test, the stomach-contents or the filtrate of stomach-contents is diluted with $\frac{1}{3}$ volume of glacial acetic acid and extracted with ether. A few cubic centimeters of this acid ether extract are mixed with 10 drops of tincture of guaiac and from 20 to 30 drops of turpentine. If blood is present, the admixture turns bluish-violet; if blood is absent, it turns reddish-brown, with a tinge of green. The reaction is still more distinct if water is added and the blue pigment is extracted with chloroform. The blue coloration of the tincture of guaiac caused by turpentine in the presence of blood is due to the action of the iron-containing constituent of blood-pigment—namely, hematin.

This test is altogether reliable, easily executed, and can, therefore, be recommended in general practice. Of course, raw or half-raw meat should not be eaten before the test is made.

The Examination of the Vomit.—The examination of the vomit frequently yields important information. The same rules that apply to the examination of aspirated stomach-contents apply to the chemical and other methods of examination of the vomit, and the same course of examination should be followed. I must emphasize that, however valuable the information may be that the analysis of the vomit reveals, it can never replace the study of the gastric secretion or the analysis of stomach-contents aspirated after a test-meal or a test-breakfast. The only method of examination that gives us positive information in regard to the chemical properties of the stomach-contents is the diagnostic aspiration of stomach-contents at a stated time after the ingestion of a test-meal or a test-breakfast. It is true that the vomit may give us some information in other respects, but in regard to the chemistry of the stomach, the results obtained from analysis of the vomit are frequently misleading. The admixture of mucus, saliva, and bile to the gastric juice obscures the results; in addition, the time elapsing between the ingestion of food and vomiting may be so short that no information whatever is obtained in regard to the functional powers of the stomach.

Some information in regard to the chemism of the stomach may occasionally be obtained from an examination of the vomit—for instance, the presence of free hydrochloric acid may be revealed or its absence may be determined. It is not well, however, in the latter instance to draw any conclusions in regard to the reduction of the secretory powers of the stomach.

The physician, at all events, should never be satisfied with the description of the vomit furnished by the patient, but should himself perform a careful inspection, followed possibly by a more exact examination.

Sometimes the vomit consists of mucus alone, of mucus and bile, or of morsels of food. If the vomit consists chiefly of morsels of food, it is interesting to know when the last meal was taken and of what it consisted. The conclusions drawn from the examination of the vomit may vary according to what is found; if, for instance, coarse morsels of food are found in the vomit six hours after the meal, a different conclusion must be drawn than if they are found immediately after the meal. We can sometimes render important decisions in regard to the character of the patient's digestive powers from the appearance of the vomit, provided we consider the time that has elapsed since the ingestion of the food. Occasionally the nature of the disease may be suspected from simple examination of the vomit. If food is vomited that was eaten the day before, we can certainly assume that the motor power of the stomach is much reduced. In many diseases of the stomach—for instance, ulcer—vomiting occurs at the height of digestion; in others—for instance, certain nervous diseases of the stomach—it usually occurs soon, often immediately after, eating. In ectasy, again, vomiting occurs at a late stage of digestion, and is usually very abundant. In a case of this kind the appearance of the vomit may vary greatly, depending altogether on the existence, for instance, of hypersecretion with ectasy, and a severe degree of gas-fermentation or of carcinomatous stenosis of the pylorus of high degree, complicated with ectasy. In the former instance the vomit will consist of abundant fluid and fine remnants of amylaceous material; in the latter we shall find coarse undigested morsels of meat. The vomit in the former case will turn Congo-paper blue; in the latter case it will not change its color.

The discovery of blood in the vomit is also important. The appearance of the blood is dependent on several conditions—on the presence or absence of hydrochloric acid, on the time that the blood has been in the stomach, on the rapidity with which it was poured into the stomach, etc.; it may look like coffee-grounds, chocolate, or fresh blood.

Pus is rarely found in the vomit. It is impossible to decide, without a careful examination directed toward other parts, whether the pus comes from a phlegmonous gastritis, from perforation of some neighboring pus focus into the stomach, or whether it has been swallowed.

Toward the end of a violent attack of vomiting bile is frequently found, but has no significance; or bile may be found in the vomit, if vomiting occurs when the stomach is empty. This finding, too, is devoid of diagnostic significance. If, on the other hand, bile is constantly vomited for a long period of time, this is an important symptom. I have described a number of cases of this kind. The vomiting of bile usually indicates that there is regurgitation of bile into the stomach, and that lesions exist that favor this abnormal occurrence. The causes may be different; there may be torsion of the first portion of the duodenum, stenosis of the duodenum by carcinoma, gall-stones,[1] etc.

A great deal of saliva or mucus may be present in the vomit; occasionally a piece of gastric tumor is found, but this is rare. Parasites, ascarides, oxyuris vermicularis, etc., are occasionally found in the vomit.

The Microscopic Examination of the Stomach-contents.—In the majority of cases the physician will be able to formulate a distinct picture in regard to the functional disturbances of the stomach with the aid of the methods we have outlined; he will be able to do this only, however, if he utilizes the results of *all* his examinations—namely, his studies of the chemical and motor functions of the stomach and of other points that we have mentioned. Microscopic examination rarely furnishes information that cannot be obtained by other methods; it may, however, corroborate and strengthen the results obtained by other examinations. I do not hesitate to say that in the majority of instances the macroscopic inspection of the stomach-contents is more important than the microscopic examination. We can frequently get along without the latter, but never without the former; at the same time I do not wish to depreciate the value of microscopic examination, and I realize that in many cases it is of fundamental importance.

The stomach-contents[2] aspirated after a test-breakfast or a test-meal or the vomit may be examined microscopically. The material aspirated from an empty stomach can also be examined in this way. The result may vary according to the food eaten or according to the time it has been in the stomach, also according to the secretory and motor powers of the organ. Microscopic examination of the aspirated or vomited stomach-contents gives us the same information as the macroscopic examination. The picture is, of course, more delicate; by this method we can readily study the more minute changes that the different articles of food, shreds of meat, amylacea, fats, etc., have undergone.

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[1] Schüle recently reported an interesting case of biliary regurgitation resulting from obstruction of the small intestine by a gall-stone, in *Berlin. klin. Wochenschr.*, 1894, No. 45.

[2] Turck (*Wien. med. Wochenschr.*, 1895, No. 1) has recently described a special method for obtaining masses of mucus that cannot be removed from the stomach by ordinary methods. He cleanses the mucous membrane of the stomach mechanically, in a manner analogous to the old method of employing a stomach-brush; he uses a small sponge that is attached to a bougie, and can rapidly be rotated within a rubber tube. I have no personal experience with this method, but it appears to me not to be devoid of danger.

Microscopic examination of the meat-fibers is of subordinate importance. If the gastric secretion is very much reduced, macroscopic examination will usually reveal a loss of peptic power if the morsels of meat are seen to be coarse and large. The microscope will reveal that the striations of muscle tissue have been preserved, but this is hardly necessary; on the other hand, we frequently see that the transverse striations of the muscles is no longer visible, even though the food has been in the stomach for some time and even though the secretion of gastric juice is altogether insufficient.

In other cases again muscle-fibers will be well digested, but the amylaceous food will not be digested; this is seen particularly in cases of hypersecretion and of great hyperacidity. Here, too, macroscopic inspection of the stomach-contents will reveal that the digestion of amylacea is incomplete. With the aid of the microscope this can be demonstrated more clearly. If a small quantity of stomach-contents is mixed with a drop of tincture of iodin or of potassium iodid solution, a large number of blue granules of amylaceous material will be seen. On microscopic examination other vegetable tissues may also be discovered.

If much fat is present in the stomach-contents, it can usually be recognized with the naked eye. The microscope reveals fat-droplets and crystals of fatty acids.

Other abnormal constituents of the stomach-contents that are seen on microscopic examination are cell nuclei, squamous epithelium, cylindric epithelium, mucus- and pus-corpuscles.

Sometimes isolated specimens of the epithelia of the gastric mucosa and of the gastric glands are seen. We possess no means of differentiating these epithelia from cancer-cells. It is of the greatest diagnostic value to find specific elements of carcinoma; single cells can never be diagnosed as cancer-cells, and the presence of cancer can be suspected only if nests of cancer-cells are seen. Ewald and others have reported findings of this character. Unfortunately, such a discovery is rare. [While the results obtained from examinations of fragments of the gastric mucosa obtained by washing the stomach are generally looked upon with doubt, Hemmeter[1] recommends a method which he says has proved successful in his hands. The stomach is thoroughly washed out at night, and the patient nourished by the rectum for a day or two thereafter. Then, without feeding, the stomach-contents is aspirated; meanwhile the tube is moved about somewhat actively in the stomach, so that the separation of particles of diseased mucosa would be favored. The wash-water is allowed to stand, and the sediment examined for diseased tissue. When pieces of mucosa show the characteristic degeneration, a strong suspicion of carcinoma is allowed.—Ed.]

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[1] *Diseases of the Stomach*, p. 526.

We refer to the statements made above in regard to the microscopic determination of the presence of blood in the stomach-contents.

The presence of micro-organisms, schizomycetes, and other forms is important. Normally isolated microbes are found in the stomach-contents, so that their presence is not pathologic; their number, however, or the preponderance of a certain species, may be pathologic. These factors will depend altogether on the presence or absence of conditions favorable for their development. The intensity of hydrochloric acid secretion, the character of the medium, etc., will determine the species that is present.

Since the proof has been furnished that the digestion of albumin does not occur in the stomach alone, but that the pancreatic ferment is capable of peptonizing albumin still more energetically than the gastric juice, the chief action of hydrochloric acid is no longer considered to be its albumin-digesting power, but its power of destroying micro-organisms. There can be no doubt that free hydrochloric acid normally impedes fermentation to a certain degree. It has been shown, however, that hydrochloric acid does not completely destroy all the germs that enter the stomach—in fact, it is known that a great development of organisms and much fermentation may occur in the stomach notwithstanding hypersecretion of hydrochloric acid. On the other hand, we frequently find that no fermentation occurs in cases where free hydrochloric acid is absent. The motility of the stomach is one of the most important factors that determine the occurrence or the absence of fermentation; in general it may be stated that fermentation develops only in cases where the motility of the stomach is reduced; in other words, where the ferment organisms can remain localized in one place for a certain length of time. If the motor power of the stomach is good, fermentation cannot occur.

Many varieties of micro-organisms are found in the stomach. Minkowski[1] has shown that if the stomach-contents contains much free hydrochloric acid, yeast and thread-fungi in particular may be found in the filtrate. On the other hand, the filtrate of stomach-contents that does not contain free hydrochloric acid usually contains numerous mold organisms. In order to determine the presence of these different germs it is necessary to allow the stomach-filtrate to stand for some time. Minkowski states that in cases in which the filtrate of stomach-contents contained no free hydrochloric acid, a great variety of fermentative and putrefactive processes developed if the filtrate was allowed to stand.

It would lead us too far to discuss all the different microbian species that are occasionally found in the stomach-contents.

The most important of the schizomycetes is yeast (see Fig. 13). If the contents of the normal stomach is examined, a few isolated yeast-cells will be found that show no evidence whatever of germination. In cases of ectasy or atony of high degree in which there is much gaseous fermentation we see an altogether different picture. Under the microscope numerous yeast-cells will be found that are arranged in colonies and that are in active process of germination; they may be so numerous that they occupy the whole field.

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[1] Minkowski, see Naunyn, *Mittheilungen aus der medicinischen Klinik zu Königsberg*, Leipsic, 1888.

Oppler[1] has shown that the significance of sarcina in the stomach-contents is the following: In benign cases of ectasy or in atony of long standing and of high degree sarcina is always present in large numbers. In acute and chronic gastritis, in atony, in peptic ulcer, in nervous diseases of the stomach, and in the dislocations of the organ the organism will only occasionally be found, usually by chance.

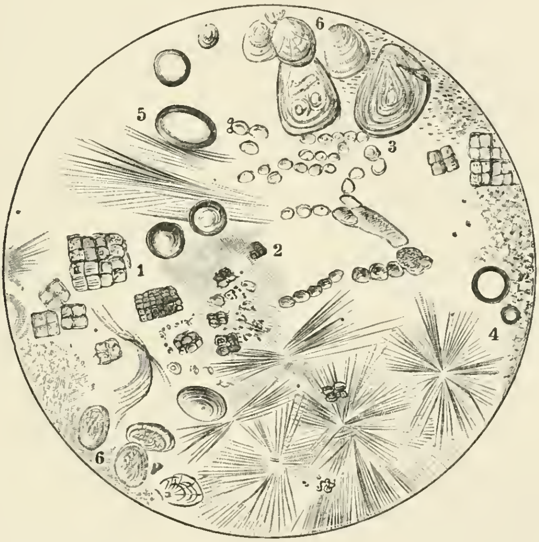

FIG. 13.—1 and 2, Sarcina ventriculi; 3, yeast; 4, needles of fatty acids; 5, fat-droplets; 6, starch-granules.

According to Oppler, sarcina is very rarely found in ectasy caused by cancer. Our own observation teaches us that sarcina is very rare in cancer of the stomach; in a few cases, however, we have seen this organism. [These conclusions are confirmed by the later observations made in this country.—ED.]

Sarcina usually appears in the shape of a bale or packet (compare Fig. 13), less frequently in the form of irregular conglomerations of individual cells. Two sizes of sarcina are found—a large one and a small one. No particular significance can be attached to the occurrence of the one or the other form. The large organism is usually smaller if it appears as an individual cell than if it is arranged in bales. It would lead us too far to discuss all the forms of schizomycetes that occur in the stomach-contents. De Bary [1] has studied the subject exhaustively, and we are indebted to him for much of our knowledge; only if stagnation of stomach-contents persists for a long time do these germs find suitable conditions for their development.

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[1] *Münch. med. Wochenschr.*, 1894, No. 29.

We will briefly discuss one species of bacillus in this place. Oppler-Boas [2] have called attention to the appearance of a peculiar bacillus in the gastric juice of carcinoma cases. This bacillus can be recognized readily in unstained specimens, and is characterized by its shape, great size, and motility (compare Fig. 14 from a case of pyloric carcinoma). According to the exact investigations of Kaufmann,[3] this organism has the power of generating lactic acid from different sugars. Kaufmann succeeded in demonstrating the presence of this long bacillus 19 times in 20 cases of carcinoma; the bacillus was absent in only 1 case, and here the absence of lactic acid was determined at the same time. It seems that large quantities of lactic acid and large numbers of these bacilli usually are observed together; Kaufmann, therefore, suggests that if this finding should be verified, it would be simpler to determine the presence of lactic acid in the stomach by examining a drop of the vomit or of the aspirated stomach-contents for this bacillus than to carry out the complicated chemical analysis of the stomach-contents for lactic acid. According to the investigations of Schlesinger and Kaufmann,[4] the presence of this bacillus indicates the existence of a carcinoma. These authors also succeeded in making pure cultures of this lactic acid bacillus. A negative result is as valid as the absence of lactic acid. Only in cases in which a stenosis of the pylorus can be diagnosed does the absence of this bacillus militate against the diagnosis of carcinoma.

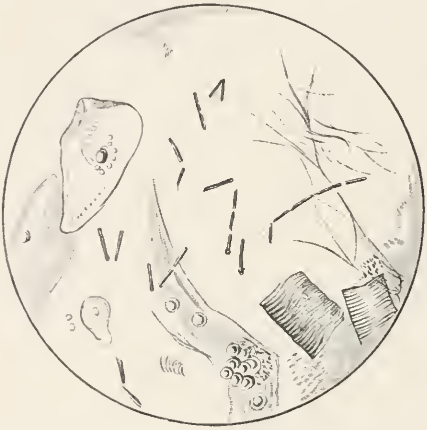

FIG. 14.—Oppler's bacilli, from a carcinomatous stomach (magnified: Seitz i. 7).

We can corroborate the occurrence of this bacillus in great numbers in the majority of cases of carcinoma. It is true, there are numerous other forms of microbes that can form lactic acid, but this does not invalidate the finding we are discussing, and although this bacillus cannot be considered pathognomonic for carcinoma, it is certainly diagnostically significant. Prazmowski[1] has studied another bacillus, the so-called butyric-acid bacillus, but it appears that this organism is not so important in the diagnosis of carcinoma as the preceding one.

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[1] *Arch. f. exp. Pathol. u. Pharmak.*, vol. xx.

[2] Oppler, "Zur Kenntniss des Mageninhaltes bei Carcinoma ventriculi," *Deutsch. med. Wochenschr.*, 1895, No. 5.

[3] Kaufmann, "Ueber einen neuen Milchsäurebacillus und dessen Vorkommen im Magensaft," *Wien. klin. Wochenschr.*, 1895, No. 8.

[4] Schlesinger and Kaufmann, "Ueber einen Milchsäure bildenden Bacillus und sein Vorkommen im Magensaft," *Wien. klin. Rundschau*, 1895, No. 15.

It has frequently been found that small fragments of mucous membrane are torn off in pumping out the stomach, however carefully the passage and the withdrawal of the stomach-tube may have been performed. As a rule, no bad consequences follow this accident. The question arises whether such an exfoliation of mucous membrane, provided, of course, that the stomach-tube is gently introduced, can be considered a sign of some pathologic process. We certainly do not feel justified in answering this question with "yes"; at the same time, we may expect to see the occurrence of this accident more frequently in cases in which there are a loosening and a tumefaction of the mucous membrane of the stomach. A careful microscopic examination of the mucous membrane that is exfoliated in this way may occasionally give some diagnostic clues. At the same time, we should be careful how we interpret these findings, particularly as the shreds of mucous lining come from the uppermost layer of the mucosa. Boas has studied a number of cases of this kind, and has shown that some diagnostic value must be attached to the microscopic examination of these shreds. Sometimes the picture of a severe or of a mild degree of interstitial gastritis will be seen, the gland-structures may be well preserved or not, the glandular epithelia may be unchanged or degenerated. From all these observations certain conclusions in regard to the character and the intensity of the disease-process may be drawn. As a rule, however, such favorable results will not be obtained, and only in exceptional cases will we be enabled to utilize these shreds of mucous tissue for diagnosis.

[In 1894 Max Einhorn described a condition which he considered a special affection, and called it "erosion of the stomach." It was characterized by finding in the wash-water aspirated from the fasting stomach small pieces of the gastric mucosa. Previous to this time similar cases had come under my own observation, and my experience in the matter is closely parallel to that of Einhorn. The patients suffered from gastric distress and sometimes from considerable inanition. The symptoms in other respects do not correspond with those of gastric catarrh, and I am satisfied that the pathology of the two conditions is not identical. Einhorn has since published the detail of 13 additional cases,[2] a number of which were cured by diet and local treatment.—Ed.]

An examination of the stomach-contents after fasting may also occasionally give some diagnostic information. Jaworski[3] first called attention to the fact that a large or small number of cell-nuclei that are arranged in the characteristic groups are occasionally seen in the stomach-contents after fasting, particularly if there is much secretion of gastric juice. These structures are formed by fragmentation of the nuclei of the leukocytes whose protoplasm has been digested by the gastric juice. The figures consist of groups of from two to seven fragments. Jaworski has also described peculiar, snail-shaped spirals that he found in an empty stomach when the secretion of gastric juice was stimulated. Boas claims that these are always found in the stomach-contents of cases in which the secretion of hydrochloric acid is normal; they are caused by a peculiar action of the hydrochloric acid on the mucus of the stomach. Their diagnostic significance is *nil.* Desquamated stomach epithelia and fragments of isolated, desquamated rennet-glands are occasionally seen in the secretion of the empty stomach. Here, too, bacteria of different kinds are often found.

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[1] Prazmowski, *Untersuchungen über die Entwicklung und Fermentwirkung einiger Bakterienarten*, Leipsic, 1880.

[2] *Jour. Amer. Med. Assoc.*, May 20, 1899.

[3] Jaworski, "Ueber die Verschiedenheit in der Beschaffenheit des nüchternen Magensaftes bei Magensaftfluss," *Verhandl. d. VII. Cong. f. innere Med.*, 1888.

In bile-tinged gastric secretions crystals have occasionally been found. Boas reports plates of cholesterin and spheres of leucin. Naunyn mentions as a rare finding numerous crystals of oxalic acid, and Eichhorst, crystals of ammoniomagnesium phosphate.

As will be seen from the above, the microscopic examination of the stomach-contents may, in certain cases, furnish valuable information. The most important microscopic findings, at all events, are particles of carcinomatous growths. The presence of numerous yeast-cells in process of germination is also diagnostically significant, whereas the discovery of isolated yeast-cells is devoid of all significance. It appears that the presence of large numbers of the long, lactic-acid bacillus is diagnostically important; all the other findings that we have enumerated are, as a rule, made by chance, and hardly merit more than passing mention. In isolated cases they may be of value in the diagnosis.

Short Scheme of the Course of a Clinical Examination.—In the preceding sections we have discussed the different methods of examination that should be employed in different diseases of the stomach. For the sake of clearness I will conclude this section by briefly enumerating the important methods in the order in which they should be employed.

1. The Anamnesis.

2. The Determination of the General Condition of the Different Organs.

3. The Physical Examination of the Stomach.

(*a*) *Inspection of the Gastric Region.*—Particular attention should be paid to protrusions or changes in the outline of the whole gastric region or of parts of this region. Attention should be given to the presence of visible tumors, of the change of position that these tumors may undergo when the stomach is filled or emptied. Peristaltic movements of the organ should be recorded.

(*b*) *Palpation of the Gastric Region.*—The attempt should be made to palpate the boundaries either of the whole stomach or of its lower portions. Tumors should be searched for by palpation, and if they are found, their consistence, their motility on respiration and on passive movements, should be studied. The presence or absence of pain and its character—that is, whether it is diffuse or circumscribed—should be determined.

Succussion sounds should be looked for, and the area determined in which they can be heard. It is important to record the time of examination—that is, whether it is carried out early in the morning, when the stomach is empty, or later in the day, at the height of digestion.

(*c*) *Percussion of the Stomach Boundaries.*

(*d*) *Inflation of the Stomach with Air or Carbon Dioxid.*—Not only the lower, but also the upper, boundary of the stomach should be determined, as well as the general outline and the position of the organ. If tumors are present, it is necessary to determine whether they change their position when the stomach is inflated or whether they can be felt better, or not so well, before or after inflation.

4. Examination of the Stomach-contents after a Test-breakfast or a Test-meal.—The stomach-contents should be examined for—

(*a*) *Quantity, Color, Odor, Appearance.*—It should be determined whether coarse particles of food are present, and what they consist of; whether there is an abundant formation of foam or development of gas; whether there are extraneous admixtures, as mucus, bile, blood, pus, etc.

(*b*) *Its Chemical Reactions.*

Course of the Chemical Examination.—*1. The Test with Congo-paper or Phloroglucin Vanillin.*—A positive reaction demonstrates the presence, a negative one the absence, of free hydrochloric acid.

2. (*a*) If the reaction is positive, quantitative determination of free hydrochloric acid with Congo-paper or phloroglucin vanillin, followed by quantitative determination of the total acidity.

(*b*) If the reactions for free hydrochloric acid are negative, determination of acid or alkaline reaction, then tests for lactic acid or for other organic acids, followed by determination of the total acidity. If it is desired to perform a quantitative determination, the hydrochloric acid deficit should be determined by adding $\frac{1}{10}$ normal hydrochloric acid solution until Congo- or the phloroglucin vanillin reaction is positive; or the combined hydrochloric acid may be quantitatively determined.

3. The Test for Pepsin.—Whether free hydrochloric acid is absent or present, it is desirable to perform an artificial digestion of a disc of albumin in the incubator in order to determine the presence or absence of pepsin in the stomach-contents. If free hydrochloric acid is absent, hydrochloric acid should be added to the stomach-contents until the Congo-reaction appears.

5. The Test for the Motor Power of the Stomach.—This is generally executed according to Leube's method. As a rule, however, the examination of stomach-contents at the height of digestion gives sufficient information for the diagnosis of the motor power of the stomach. In cases of atony, or if hypersecretion is suspected, the stomach-contents should be examined early in the morning, before breakfast; in the former case after the administration of a simple supper; in the latter case after a long period of fasting, or after the stomach has been thoroughly washed out and emptied the evening before.

6. The Microscopic Examination of the Stomach-contents.—If carcinoma is suspected, numerous long bacilli should be looked for. If there is much gaseous fermentation, large numbers of yeast-cells should be looked for.

This is the ordinary course of an examination of the stomach and its contents. It should be carried out in every case. Certain special methods—as, for instance, the fermentation test, the examination of the stomach-contents for blood, etc.—may be indicated in individual cases. In the majority of instances the above-named methods will be sufficient.

THE EFFECT OF DISEASES OF THE STOMACH ON OTHER ORGANS.

In the preceding sections we have limited ourselves to a discussion of the methods for examining the diseased stomach, and have simply described the pathologic findings in cases of this kind. In practice, however, more is necessary: it is not enough to limit the examination to that organ that is the primary seat of the trouble: it is imperative that all other organs should be examined. This applies to disease of any organ, and particularly of the stomach, for we know that no other organ is capable of affecting other remote organs to such a degree as the stomach when it is diseased, and inversely we know that diseases of many organs seem to have a particular tendency to affect the stomach secondarily.

It will, of course, be impossible to discuss all the remote effects of diseases of the stomach on other organs in this place, and we will limit ourselves to a brief mention of a few of the most important and most frequent of these remote effects.

Diseases of the stomach, of course, primarily influence general nutrition; in the second place, they affect the action of the heart; finally, they exercise a decided influence on the nervous system. The urine and the blood also show changes in diseases of the stomach that are diagnostically significant and may be briefly mentioned.

Influence of Diseases of the Stomach on the General Nutrition.—Most cases that are suffering from disease of the stomach state that they began to emaciate after the disease has persisted for some time. In a small minority of cases the general nutrition is not impaired; for this reason it is necessary that the physician should determine how mach flesh the patient has lost and within what time the loss of flesh occurred. It certainly makes a difference whether the patient maintained his weight notwithstanding the existence of gastric trouble for a period of years, or whether he had lost ten or more kilos within a few months.

We shall not be surprised to learn that in acute or very serious diseases of the stomach that are complicated by violent and frequent vomiting, and in which consequently the ingestion of food is rendered difficult, nutrition is impaired, nor will we be surprised if general nutrition is impaired in a number of chronic diseases of the stomach. In many cases we will be able to see at once that the nutrition of the patient is below par. It is impossible, of course, to determine whether the emaciation that we see is due to a deficient ingestion of food or whether other factors are concerned in this process. The only way to decide this question is to make careful studies of the general metabolism of the patient.

If general nutrition is reduced, the first task of the physician is to raise it. In order to do this intelligently he must first determine whether or not the patient is taking too little food, and if so, why. We cannot say in every case that the cause of impaired nutrition is the ingestion of too little food.

A healthy subject does not need to take his weight at regular intervals, nor need he calculate how many calories he must introduce daily in order to maintain his weight, nor finally does he have to know that a healthy human being needs from 30 to 40 calories pro kilo of his body-weight *pro die*, and that the number of calories that he must introduce will depend on the amount of exercise that he takes. Although he pays no attention to these questions, he succeeds in maintaining an average uniform weight. A healthy subject introduces only so much food as he is capable of oxidizing. The number of calories that are needed for the maintenance of his weight are regulated by his appetite and by his desire for food. In diseases of various kinds this is not the case, particularly not in diseases of the stomach; here the regulator is disordered, the appetite of the patient no longer corresponds to the amount of nourishment that the organism calls for. In a healthy subject the appetite is the best indicator of the amount of nourishment that is needed. In certain diseases we can no longer control this factor through the appetite of the patient; this does not only apply to diseases of the stomach, but applies with equal force to acute febrile diseases in which this regulator is without exception disturbed. In fever the consumption of material and the demand for it never equalize each other, and consequently we see such extreme degrees of emaciation in febrile diseases, particularly as there is a general prejudice against administering an abundant diet in these conditions.

In many cases of stomach-disease there is lack of appetite or even repulsion for food, and this leads to a deficient introduction of nutritive material. The disease may be a serious one, on the ground of these subjective dislikes, and the loss of strength may be due to this factor alone. This lack of appetite is seen in acute and in chronic diseases of the stomach—for instance, in acute and chronic catarrh, in carcinoma, and in many forms of nervous dyspepsia.

In other cases the patients do not like to eat because the swallowing of food causes pain. In many instances these patients have a good appetite, but they do not dare to eat on account of pain; this is particularly the case in gastric ulcer and in many forms of hyperacidity.

In other cases, again, vomiting occurs after eating, so that the patients eat as little as possible. Occasionally the physician is to blame, for many practitioners are in the habit of treating the stomach by attempting to spare it by giving little food, without considering the individuality of the case and without prescribing food that contains a sufficient number of calories. Many physicians nowadays give some very general directions in regard to the diet, or content themselves with recommending a non-irritating diet without indicating the quality and quantity of the food and drink. No wonder that cases that are treated in this way emaciate and lose strength !

In acute diseases of the stomach the loss of weight is not so important, and in many of these cases total abstinence is indicated. If nutrition becomes too much impaired, rectal alimentation may be attempted in order to compensate the lack of food.

In the majority of cases inanition is not severe, and the decrease in the amount of food taken is not the exclusive cause of emaciation. Without doubt we shall find that in many of these instances the absorption and assimilation of the food are also impaired ; and, finally, in many cases of acute dyspepsia we shall see that the disturbed functions of the stomach lead to the formation of toxic substances ; these in their turn produce autointoxication and an increased proteid katabolism.

The chronic diseases of the stomach are of great practical significance. If nutrition remains impaired for too long a time, other serious consequences will result. In many, if not in all, of the cases of this character we are justified in stating that the cause of the emaciation and of the loss of strength is exclusively due to the deficient amount of food that the patient eats. Von Noorden[1] performed a number of interesting experiments in order to determine how much certain cases of stomach-trouble would eat if they were allowed to follow their own inclinations. He instructed his patients to write down every two or three days what they had eaten of their free will. In this way he determined that in cases of moderate chronic disturbances, as ulcer, chronic catarrh, and nervous dyspepsia, an average of 21 calories pro kilo was ingested. This means that the ingestion of food was reduced to one-half of the necessary quantity, and it is not surprising, therefore, that these cases suffered from great impairment of nutrition and great loss of strength. We know that the caloric value of the food that an adult should eat in order to maintain his body-weight is equal to from 30 to 40 calories *pro die* and pro kilo of body-weight when the patient is at rest ; and from 40 to 45 calories when he is doing a moderate amount of work.

Another question seemed particularly interesting—namely, to determine to what extent the food was assimilated in cases where the function of the stomach was reduced or where there was great subacidity. Von Noorden's[2] metabolic studies in this direction have furnished some very valuable information ; he examined cases in which the secretion of hydrochloric acid was so slight that only a small portion of the albumin was peptonized in the stomach, and the greater portion passed into the intestine in an undigested state. He found that the assimilation of the chief articles of diet was not reduced, even though the peptic powers of the stomach were poor. He showed that the intestine can act vicariously for the stomach in cases where there is such a deficit of hydrochloric acid in the stomach that the proteids cannot be digested there. This occurs, however, only in cases in which all the food enters the intestine without being decomposed; if decomposition occurs or if the food is retained in the stomach too long, so that it undergoes fermentative changes, the absorption and the assimilation of nutritive material are reduced. This is seen in cases of subacidity complicated by atony.

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[1] Von Noorden, "Ueber den Stoffwechsel der Magenkranken und seine Ansprüche an die Therapie," *Berlin. Klinik*, No. 55.

[2] *Zeitschr. f. klin. Med.*, 1890, vol. xvii.

Experiments that have been made in animals yield the same results. If the stomach is ligated in animals, the same metabolic changes are observed as in human beings when the gastric juice is subacid or anacid. Here, too, it was found that the cessation of gastric digestion did not interfere with general nutrition, so that the conclusion can be drawn that the function of the stomach can be dispensed with, at least in regard to the preparation of the food ingested for intestinal digestion. The clinical observations are, of course, more important than the animal experiments recorded.

Although we know, therefore, that in the majority of diseases of the stomach and even in many cases of subacidity or anacidity of the gastric juice metabolic perversions indicate nutritive disturbances (von Noorden), we are never certain whether or not other factors besides gastric disease are concerned in the loss of strength; in fact, we may assume that in many cases disturbances of the gastric function lead, for instance, to the formation of toxic substances that are absorbed, injure the tissues of the body, and induce increased proteid katabolism. Although much remains to be done in this direction, we know to-day that in certain diseases of the stomach toxic substances are, in fact, formed that exercise a deleterious influence on the protoplasm of the cells. Müller[1] showed that in a number of cases of carcinoma there was an increased excretion of nitrogen that was not in proportion to the amount of nitrogen ingested. He was unable to explain how this occurred; it is probable that abnormal products were formed that intoxicated the organism and caused an increased katabolism of proteids. The same applies to certain forms of acute gastritis and some forms of ectasy that are complicated with much fermentation. That this actually occurs has never been demonstrated experimentally. In the majority of diseases of the stomach, therefore, we must assume that the emaciation is due to deficient nutrition. This fact is an important one in practice.

The general practitioner, of course, cannot perform complicated metabolic studies of his cases; nevertheless, he should determine whether sufficient food is taken or not and whether general metabolism corresponds to the amount of food ingested. In order to do this, he must observe the fluctuations in the weight of the patient, and he must consider the caloric value of the food. The diet should be chosen to fit the nature of the disease and the character of the gastric secretion. All these factors must be considered in order to be able to prescribe a sufficient diet and to counteract the disease process in a rational manner. In any form of stomach-disease the diet should have as high a caloric value as possible and should be small in bulk; at the same time it should not be irritating, chemically or mechanically.

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[1] *Zeitschr. f. klin. Med.*, vol. xvi.

In a subsequent section we will discuss the best manner of fulfilling all these conditions. In this section we simply wish to call attention to the influence of gastric diseases on general nutrition. However important it may be to spare the diseased organ, the physician should never forget that one of the chief indications of treatment is to maintain general nutrition.

The Effect of Stomach-diseases on the Action of the Heart.—There is a general belief to the effect that diseases of the stomach exercise no appreciable influence on the action of the heart. This, however, is not the case, as will be seen on careful observation. Even in a healthy subject the ingestion of a meal influences the heart-action by increasing the rapidity of the pulse. In inanition the pulse-rate is reduced, although only to a slight degree. In the well-known case of Cetti the pulse, during almost complete inanition, was reduced to only 64 beats to the minute.

Diseases of the stomach proper may either increase or decrease the pulse-rate—more frequently they do the latter.

An increase of the pulse-rate is seen particularly in those diseases of the stomach that are complicated by fever—for instance, in acute toxic or infectious gastritis, in phlegmonous gastritis, and in diseases of the stomach that are complicated, for instance, by circumscribed or general peritonitis.

We will not discuss those cases in which the acceleration of the heart-beat is proportionate to the fever. The majority of stomach-diseases run their course without fever. An increased pulse-rate is rarely seen without fever; it is occasionally observed in instances where the patients are exceedingly weak or in a state of collapse.

Bradycardia is occasionally seen. This finding is exceedingly interesting. A number of cases of reduction of the pulse-rate in diseases of the stomach are chronicled in the older literature. Only recently, however, have certain authors called attention to the connection between the two. Wagner,[1] for instance, recently described a case of dilatation of the stomach from the clinic at Leipsic in which the temperature was very low (34.5°–36° C.—94°–96.8° F.) and the pulse-rate fell as low as 44 and 40. Wagner does not see any connection between the reduction in the pulse-rate and the disease of the stomach, but declares bradycardia to be a result of the low temperature.

Ozanam[2] expresses the opinion that in certain diseases of the stomach the weakness of the patient and the vomiting lead to such a degree of exhaustion that the pulse-rate is reduced, and that in certain nervous diseases complicated with dyspepsia the pulse is slowed by reflex action through the vagus nerve. The same, he thinks, applies to cases of severe indigestion with vomiting. In cases of the latter kind he claims to have observed a great reduction of the pulse-rate for several days ; at the same time he thinks that such an occurrence is exceptional.

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[1] See "Berichte d. med. Gesellschaft zu Leipzig, Sitzung von 11. Mai, 1880," *Berlin. klin. Wochenschr.*, 1881, No. 16.

[2] Ozanam, *La circulation et le pouls, histoire, physiologie, sémiotique, indications thérapeutiques*, Paris, 1886.

Grob[1] has published a very careful dissertation on bradycardia. He mentions the reduction of the pulse-rate in diseases of the stomach, but states that this occurrence is very rare. I[2] have expressed a different opinion in a report published some time ago, and have stated that a reduction in the pulse-beat is not rare in diseases of the stomach. In fact, I have shown that in stomach-affections a particularly low pulse-rate is often seen. In cases of ulcer, ectasy, and other diseases of the stomach I have quite frequently seen the pulse beat only 44, 42, or 40 times a minute. In the case of a girl of nineteen who was suffering from a mild degree of chlorosis with dyspeptic symptoms, we saw bradycardia for three weeks in which the pulse beat only 34 times a minute.

Before discussing the frequency of bradycardia in diseases of the stomach I wish to refute an argument that might be adduced against a connection between the two conditions. It might be argued that bradycardia, when it is seen in combination with diseases of the stomach, is not a result of this disease, but an exceptional complication, or the result of a disease of some other organ. This argument, however, is refuted when we consider that, as a rule, bradycardia ceases as soon as the condition of the stomach improves. At the same time the persistence of bradycardia after the cessation of stomach-symptoms would not prove that the former was not dependent on the latter. An ulcer of the stomach, for instance, cannot be considered cured even if the chief symptoms, as cardialgia, vomiting, etc., disappear, so that even in cases where the main symptoms of the stomach-disease disappear, we need not necessarily expect that the bradycardia will disappear.

According to my own observations, I believe that ectasy and ulcers of the stomach are most frequently followed by a reduction in the pulse-rate. Bradycardia, it is true, is seen in a variety of other diseases of the stomach, but not so frequently and so constantly as in the above-named affections. It appears that bradycardia follows only serious diseases of the stomach, and that the two are intimately connected ; we are unable to give any reason for this.

At the same time there are a number of physiologic facts that demonstrate the intimate connection between the stomach function and the action of the heart. It is well known that different gastric irritants may cause a reduction of the pulse-rate and an increase of arterial pressure by sending stimuli along the vagi. We also know by physiologic experiments that inflation of the stomach or any tension of the organ leads to a reduction in the pulse-rate in animals; this finding certainly throws some light on the relative frequency of bradycardia in ectasy of the stomach. It is further known that irritation of the sensory nerves of the stomach always leads to a reduction in the frequency of the heart-beat, and this is significant in interpreting the bradycardia seen in ulcers of the stomach. It is also known that the pulse-rate is reduced and that the heart-beats stop if one vagus is severed and its central end irritated, while the other vagus remains intact.

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[1] Grob, "Ueber Bradycardie," *Deutsch. Arch. f. klin. Med.*, vol. xlii.

[2] Riegel, "Ueber Verlangsamung der Schlagfolge des Herzens," *Zeitschr. f. klin. Med.*, vol. xvii.

All these physiologic facts can be utilized to explain the occurrence of bradycardia in diseases of the stomach. This symptom is important from a practical point of view, because it shows that diseases of the stomach may seriously affect remote organs.

Occasionally irregularity of the heart-action is seen in diseases of the stomach. This may occur alone or together with bradycardia. In the latter case bradycardia may be due to the fact that some of the contractions of the heart are incomplete and are not felt at the apex or at the radial artery, so that, in fact, no *real* bradycardia exists. This form of pseudobradycardia usually appears as allorrhythmia or bigeminy of the heart, and must be distinguished from true bradycardia. In diseases of the stomach arhythmia of the heart is the most frequent complication. Occasionally this condition of the heart is not produced by a disease of the stomach, but by some other cause; the symptom is intensified, however, by the stomach-affection. Arhythmia is rarely the direct result of stomach-disease; the condition of arhythmia is so common, in fact, that it is naturally also seen in many cases of stomach-disease. It will always be a difficult matter to prove in any one given case that arhythmia is the direct result of some disease of the stomach.

The Effect of Diseases of the Stomach on the Nervous System.—It is well known that cases of chronic disease of the stomach frequently complain of a variety of nervous disturbances. The patients are frequently morose, they have no desire to work, they complain of headache, insomnia, and a great variety of other nervous symptoms; in fact, the patients may become very neurasthenic, so that it may be a difficult matter to determine whether the disease of the stomach is the primary lesion or whether it is only one of several symptoms of a primary neurasthenia.

One of the most important nervous symptoms seen in diseases of the stomach is the so-called stomach vertigo, which was first described by Trousseau.[1] This condition is mentioned in some of the older works, and is called "vertigo per consensum ventriculi." The symptom may appear at different stages of digestion—either at the height of digestion, in the night, or when the stomach is empty. Trousseau mentions that the vertigo appears more frequently when the stomach is empty than when it is full. The clinical picture of this condition varies; the patients may suddenly become dizzy without feeling any pain, or there may be a little nausea and a tendency to vomit. An attack of this kind may last for varying periods of time—sometimes only for a few minutes, in other cases for a long time. Trousseau remarks that this form of gastric vertigo is never accompanied by loss of consciousness; there are, however, a few cases on record in which there was a transitory loss of consciousness. Attacks of vertigo of this kind are not so rare as is usually believed. They are more frequently seen in anemic, weak, and nervous subjects than in robust, strong individuals; they are, however, occasionally seen in the latter.

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[1] *Med. Clinic of the Hôtel-Dieu*, Paris, 1868, vol. iii., p. 1.

I have observed these attacks most frequently in cases of hyperacidity when the stomach was empty. That these attacks are directly related to the stomach is demonstrated by the fact that they disappear as soon as the patient eats something. Trousseau mentions that a cup of bouillon or a cracker dipped in wine frequently cuts short the attack or stops it after it has set in. In other cases vomiting occurs and the attack stops.

Leube[1] reports an interesting case of gastric vertigo. This occurred in a patient of forty-nine who had been a sufferer from dyspeptic symptoms and attacks of vertigo for nine years. The dizziness never occurred in the morning, but always after a meal, particularly in the evening and when the patient was sitting down; the patient would have to rise in order to get rid of the disagreeable sensation. As soon as the patient could belch, the attack of vertigo disappeared. The patient also complained of vertigo if he was lying down and something pressed on the region of the stomach.

There is a great diversity of opinion in regard to the cause of these attacks, and it is not decided whether they are a result of circulatory disturbances or whether they are caused by reflex action. Some authors have attempted to explain them by intoxication. They certainly are in some way connected with the function of the stomach; this is demonstrated by the good effects that follow treatment directed toward the disease of the stomach. [Cases of so-called typical gastric vertigo not infrequently depend upon uncorrected eye-strain, especially upon anisometropia. It is true that the attacks are often precipitated by transient disturbances in digestion, and these digestive derangements, in turn, may be occasioned by some eye-strain that predisposes to the vertigo. In several instances I have seen the disappearance of both vertiginous and gastric symptoms follow a careful and painstaking correction of the refractive error.—Ed.]

There may also be attacks of spasm, so-called tetany, that are more important than the above-mentioned attacks of vertigo; these are seen in a variety of diseases of the stomach, particularly in ectasy with hypersecretion. Kussmaul[2] was the first to call attention to the occurrence of convulsive attacks in dilatation of the stomach in his work, *On the Treatment of Dilatation of the Stomach by a New Method—the Stomach-pump.* He describes attacks of tonic spasms of the arms and legs that may involve the muscles of the body, of the face, or even the muscles of the larynx, etc. We cannot describe these tetanic spasms in this place. They do not seem to correspond exactly with the picture of tetany proper. They may be so severe as to endanger life; Kussmaul described a case that died. This author is inclined to attribute these attacks to the concentration of the blood or to the withdrawal of water from the nerves and the muscles.

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[1] Leube, *Von Ziemssen's Handb. d. spec. Pathol. u. Therap.*, vol. vii.

[2] *Deutsch. Arch. f. klin. Med.*, vol. vi.

Other authors have reported similar observations—von Gassner,[1] Leven,[2] Gaillard,[3] Dujardin-Beaumetz and Oettinger,[4] Martin,[5] Dreyfuss-Brissac,[6] Macall,[7] Renvers,[8] Müller,[9] Gerhardt,[10] DeBeurmann,[11] Merlin,[12] Loeb,[13] Bouveret and Devic,[14] Blazicek.[15]

Richartz[16] has reported 2 cases of this kind from my clinic. I myself observed a case not long ago.

To judge from the descriptions of all the authors named there can be no doubt that a symptom-complex is observed that is either identical with tetany or at least very similar to it. A number of the more important symptoms of tetany can usually be seen—for instance, Trousseau's sign, the facialis phenomenon, an increase of mechanical irritability, and Erb's increase of the electric irritability of the nerves and muscles. In some of the cases the symptoms were not looked for; in others again they were looked for and not found.

Among 27 cases that I find reported in the literature, 16 terminated fatally. Of the 2 cases reported from my clinic, both died. A case that I observed recently, in which there was a carcinoma of the stomach causing stenosis and ectasy, also died. The occurrence of attacks simulating tetany in diseases of the stomach must, therefore, be considered a very dangerous complication.

Nothing has so far been found postmortem to explain these attacks of tetany. In all cases there was ectasy, usually of high degree; generally ectasy was caused by a stenosis of the pylorus or of the duodenum; most frequently by an ulcer or a cicatrix caused by an ulcer. In several of the cases—for instance, in 2 of my own—there was a carcinoma that developed from a scar. Blazicek reports a case in which the ectasy of the stomach was caused by compression of the duodenum from pressure by the gall-bladder, which was chronically inflamed and filled with concretions. In other cases again, as in those of Bouveret and Devic, there was ectasy of the stomach with hypersecretion, so that these authors attach particular importance to hypersecretion as a possible cause of the spasms. This view is hardly tenable, for we know that tetanic attacks are often seen in cases of ectasy that are due to some other cause.

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[1] Thèse de Strasbourg, 1878.

[2] Leven, *Traité des mal. de l'estomac*, 1879.

[3] *Gazette des hôpitaux*, 1883.

[4] *Union médicale*, 1884, Nos. 15, 18.

[5] *Lancet*, 1887.

[6] *Gaz. hebd.*, 1885.

[7] *Glasgow Med. Jour.*, 1871.

[8] *Berlin. klin. Wochenschr.*, 1888.

[9] *Annales de la Charité*, 1888, vol. xiii.

[10] *Berlin. klin. Wochenschr.*, 1888, No. 4.

[11] *Société méd. des hôpitaux de Paris*, March, 1889.

[12] *Loire médicale*, 1890, No. 15.

[13] *Deutsch. Arch. f. klin. Med.*, vol. xlvi.

[14] Bouveret and Devic, "Recherches cliniques et experimentales sur la tétanie d'origine gastrique," *Revue de méd.*, 1892.

[15] *Wien. klin. Wochenschr.*, 1894, Nos. 44, 46, and 48.

[16] *Inaug. Diss.*, Giessen, 1893.

No satisfactory explanation, therefore, has so far been furnished for the origin of tetany in dilatation of the stomach. Kussmaul, as we have said, attributes tetany to the loss of water and the consequent desiccation of the tissues, and sees an analogy between these spasms and the spasms of cholera Asiatica and nostras. The tissues are undoubtedly dry in many of these cases, as they are deprived of much water. There is, in fact, a great similarity between cholera and the disease under discussion in regard to this one point, but we must never forget that it is not proved in cholera that the spasms are caused by desiccation of the tissues, for we see muscular spasms in cholera sicca, and here there is no desiccation of the tissues. Again, a number of cases of tetany have been observed in dilatation of the stomach in which examination of the blood failed to reveal a reduction of the water (see case 1 of Blazicek). This theory, therefore, has been abandoned.

Another theory claims that the tetany spasms are due to some reflex action. In support of this view the occurrence of tetany in helminthiasis is mentioned: I[1] have observed a case of this kind. In the last case of tetany that I observed (see above) the attacks followed the introduction of the stomach-tube. Müller has also described a case in which tetany developed whenever the region of the stomach was tapped. These observations all speak in favor of the reflex theory; at the same time the theory is not satisfactory, for it does not explain why tetany is absent in certain forms of ectasy.

A third theory that is in the field is more satisfactory; it attributes the spasms to autointoxication from the intestinal tract. Some authors speak in a general way of the absorption of poisonous substances formed by the decomposition of stagnating stomach-contents. Others again, as Bouveret and Devic, claim that the hypersecretion of the stomach and the formation of peptotoxins in the presence of free alcohol is the most important factor. This theory is not proved, even though Bouveret and Devic succeeded in producing spasms resembling tetany in animals by the intravenous injection of an artificial mixture of albumin, free hydrochloric acid, and alcohol. Besides, the cases of hypersecretion mentioned above do not agree with the theory of Bouveret and Devic. It would lead us too far to discuss this question at greater length.

Certain disturbances of the sensorium—coma, stupor, and delirium—are occasionally seen in diseases of the stomach, not only in the above-named cases of tetany, but also in the later stages of carcinoma. Here, too, the theory seems plausible that toxic substances are absorbed.

Asthma dyspepticum also belongs to this category. This complication is rare; it was first described by Henoch[2] and later by Silbermann[3] and others. It is questionable whether the name asthma dyspepticum is happily chosen, for the symptom-complex of this form of asthma does not correspond to the picture of nervous asthma in the true sense of the word.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xii.

[2] *Berlin. klin. Wochenschr.*, 1876, No. 18.

[3] *Ibid.*, 1882, No. 23.

Henoch believes that the syndrome seen is the result of reflex action starting from the stomach. He adduces as an argument in favor of this supposition the observation he made in 2 cases—namely, that the more severe symptoms disappeared when the patient vomited. The syndrome of this complication is as follows: The breath is rapid and shallow, there is cyanosis, the pulse is small and rapid, the extremities are cool, the temperature is subnormal, and there may be symptoms of collapse. By far the greatest number of cases of dyspeptic asthma have been observed in children.

In all cases the breathing was rapid and dyspneic, although an examination of the thoracic organs failed to reveal any lesions. The region of the stomach was usually distended and painful, the extremities were cool, the face cyanotic, the pulse small and frequently so rapid that its beats could not be counted. In all cases there was acute dyspepsia as a result of some error in diet. The disease never lasted longer than one day.

Henoch utilizes Mayer-Pribram's observations in dogs to explain the occurrence of asthma dyspepticum. The latter experimenter saw that in dogs and cats an increase of the arterial pressure and a slowing of the pulse occurred if the stomach was irritated in different ways. Henoch believes that a reflex stimulus starts from the stomach and causes the vasomotor spasm in the smaller arteries. As a result, the extremities feel cool, the pulse is small or imperceptible, there is stasis in the venous system and the right heart, cyanosis, an accumulation of carbon dioxid in the blood, and consequently dyspneic breathing. Silbermann accepts this explanation for some of the cases, but not for all. He believes that paralysis of the heart may be the primary factor, and that this may lead to an engorgement of the lesser circulation and of the right heart, followed by rapid breathing and dyspnea.

There may be a diversity of opinion, as I have already said, in regard to the advisability of calling this symptom-complex asthma. The condition does not correspond to cardiac asthma, and is distinguished from this disease by the rapid breathing. It is well known that acceleration of the heart-action and of breathing occurs more readily in children than in adults, and the majority of cases of dyspeptic asthma are seen in children. The fluctuations in the number of respirations and the rapidity of the pulse, as we know, increase with the age of the patient; in adults I have frequently seen dyspnea, oppression, and terror as a result of diseases of the stomach, but never attacks that corresponded to asthma proper. Many of the cases described by other authors are doubtful in the same sense. It is true that the breathing may be accelerated and the pulse may be weak in cases where there are great distention of the stomach and pain in the gastric region, and that these symptoms may frequently recede as soon as vomiting occurs; but an attack of this kind certainly is not an attack of asthma.

Although I am unable, therefore, to concede that the symptom-complex described above is true asthma, and although it is exceedingly doubtful whether the attacks are due to reflex action, I still feel that they should at least be mentioned in this place.

In the preceding paragraphs I have discussed a number of disturbances of the nervous system and of the circulatory apparatus that are occasionally seen as a result of diseases of the stomach. There can be no doubt that these disturbances are in some way connected with disturbances of the stomach function. Until very recently all authors were inclined to attribute these symptoms to reflex action; within the last few years, however, the majority of investigators attribute them to autointoxication. The most modern view is, therefore, that certain poisonous metabolic products are developed in the course of certain diseases of the stomach and intestine, and that they are absorbed and cause the disturbances described above.

It would lead us too far to discuss all the arguments *pro* and *contra* this view. I refer those of my readers who are particularly interested in this question to a monograph by Albu,[1] in which all the investigations in this field are collected. However fascinating and probable the theory is that certain nervous symptoms that are seen in the course of stomach-diseases, particularly headache, vertigo, coma, spasms, etc., are due to an autointoxication, we must not forget that no positive proof for this assertion has so far been furnished. Neither chemical examinations nor animal experiments have demonstrated the truth of this hypothesis. Even the experiments of Bouveret and Devic in the case of tetany can hardly be considered altogether valid and free from objection.

From a clinical point of view this autointoxication theory is a very probable one, for we frequently see a series of nervous symptoms following acute dyspepsia that is caused by the ingestion of improper or decomposed food. In chronic dyspepsia the matter is somewhat different; here the nervous symptoms are irregular; and they appear at different times of the day with different severity. Some appear when the stomach is full, others when the stomach is empty, so that we cannot deny the possibility of a reflex origin. For some symptoms autointoxication is undoubtedly the best explanation, even though a positive proof for this assumption is still forthcoming. We are not justified, therefore, as many authors seem to think, in attributing all nervous symptoms that are seen in the course of diseases of the stomach to the action of poisons—to autointoxication.

THE URINE IN DISEASES OF THE STOMACH.

The urine has been investigated by many clinicians in pathologic conditions of the stomach, and a great deal of interesting information has been obtained; nothing, however, of practical value has so far been discovered. It may be interesting, at the same time, to mention briefly the most important points.

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[1] Albu, *Ueber die Autointoxicationen des Intestinaltractus*, Berlin, 1895.

In regard to the quantity, we find no abnormalities in stomach-diseases, provided that the ingestion of food, the gastric digestion, and absorption are approximately normal. The same applies to the specific gravity of the urine. If the amount of food taken, however, is insufficient, or if there is much vomiting, or, again, if the motor power of the stomach is reduced, the quantity of urine excreted decreases. This is seen particularly in cases of ectasy of the stomach of high degree, sometimes only 400 or 500 c.c. being excreted. Such a reduction is, of course, very significant, and an increase of the flow of urine under these conditions must be considered a good prognostic sign.

In regard to the reaction of the urine we know that it is uniform, as in the case of the other secretions and excretions, but that it depends to a certain degree on the secretory activity of the stomach. Bence Jones[1] in 1819 first called attention to the fact that the acidity of the urine is reduced after eating, that the urine gradually becomes neutral or even alkaline (three hours after breakfast, five to six hours after dinner), and finally becomes acid again. Bence Jones explains this phenomenon by assuming that a certain amount of acid is withdrawn from the blood when hydrochloric acid is secreted in the stomach. Roberts,[2] on the other hand, explains the phenomenon by assuming that a certain quantity of alkali is ingested with the food and enters the blood.

Bence Jones's view was corroborated and amplified by later observations and experiments by Quincke,[3] Stein,[4] Maly,[5] and Görges,[6] and further by experiments carried on in my clinic by Sticker and Hübner.[7] In order to determine the relationship between the gastric secretion and the reaction of the urine, Sticker and Hübner performed some experiments in cases of inanition, and found that no change in the reaction of the urine occurred at that period of the day in which, during the normal ingestion and digestion of food, the absolute acidity of the urine was apt to fluctuate. It was also seen that the acidity of the urine was not reduced if accumulation of hydrochloric acid did not follow the ingestion of food into the stomach. From these experiments the conclusion can be drawn that in those cases where the acidity of the urine is not reduced after a meal (provided, of course, that some other extraneous cause cannot be made responsible), there is a decrease or a complete cessation of hydrochloric acid secretion; in fact, the degree of reduction of urinary acidity is to a certain extent an indicator of the amount of acid produced in the stomach during the period of digestion.

As the midday meal is usually the largest meal of the day, the acidity of the urine is decreased principally in the late hours of the afternoon; after supper the decrease in its acidity is not so apparent.

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[1] *Philosophical Trans.*, 1819, p. 235.

[2] *A Practical Treatise on Urinary and Renal Diseases*, 1872, second edition, p. 48.

[3] *Correspondenzbl. f. Schweizer Aerzte*, 1874; *Zeitschr. f. klin. Med.*, 1884, vol. vii., supplement.

[4] *Jahresb. d. Thierchemie*, vol. iv., p. 241, and *Arch. f. klin. Med.*, 1876, vol. xviii.

[5] Liebig's *Annalen*, vol. clxxiii., p. 227; Hermann's *Handbuch d. Physiol.*, 1881, vol. v., pt. ii.

[6] *Arch. f. exper. Pathol.*, vol. xi.

[7] *Zeitschr. f klin. Med.*, vol. xii.

Sticker and Hübner's discovery that the fluctuation in the acidity of the urine is not seen in inanition is readily explained from the physiologic relation that is known to exist between the secretion of acid into the stomach and the reaction of the urine. It will also be readily understood why the reduction of urinary acidity is not seen in carcinoma, where there is no secretion of hydrochloric acid in the stomach; why abundant vomiting of stomach-contents containing much hydrochloric acid reduces the acidity of the urine for many hours; and why, finally, the reduction in the acidity of the urine is proportionate to the production of gastric juice and decreases as the hydrochloric acid secretion increases.

The Chlorids.—The amount of chlorids present in the urine is primarily dependent on the amount of food ingested. In severe diseases of the stomach in which little food is eaten, particularly in ectasy of high degree following stenosis of the pylorus, the amount of chlorid of sodium excreted is very much reduced. If in a case of this kind the chlorin increases in the urine, this is a good prognostic sign; it demonstrates far better than increased diuresis that food is again being assimilated (von Noorden).[1] Under certain conditions the excretion of a small amount of chlorid of sodium in the urine may be utilized in combination with nitrogen determinations to find whether dilatation is complicated by carcinoma or not. If in an emaciated sufferer from some stomach-disease very little sodium chlorid and very little nitrogen are excreted *pro die*, this indicates simple inanition; if, on the other hand, there is very little sodium chlorid excretion and comparatively much nitrogen excretion, this demonstrates that there is a morbid increase in the metabolism of organic albumin that contains little sodium chlorid; this is never seen in simple inanition, but is frequently observed in carcinomatous cachexia (von Noorden). [The same observation applies in the cachexia of late syphilis.—Ed.]

Rosenthal[2] states that in certain forms of hyperacidity that are caused by mental overexertion, by violent emotional disturbances, or by migraine (frequently complicated with cardialgia and vomiting) there is a considerable decrease in the excretion of the chlorids of the urine and, at the same time, an increase of the earthy phosphates. Gluzinski and Jaworski,[3] in 1884, saw complete disappearance or a great reduction of the chlorids and of the acid reaction of the urine in cases of dilatation of the stomach with continuous hypersecretion of acid gastric juice. The experiments of Sticker[4] demonstrate that an abundant secretion of hydrochloric acid causes a decrease of the chlorids in the urine, provided that the conditions are favorable for its retention in the stomach or its excretion from the organism. Gluzinski[5] arrived at analogous results, and found that a decrease in the chlorids of the urine in stomach-diseases is seen—(*a*) if too small a quantity of chlorids is introduced (inanition, frequent vomiting immediately after eating); (*b*) if the introduction of chlorids is sufficient, but their absorption is interfered with—as, for instance, in ectasy of the stomach of high degree caused by carcinomatous stenosis of the pylorus; (*c*) if there is an excessive secretion of gastric juice, but only then in these cases if the excess of secreted hydrochloric acid is expelled by vomiting or by frequent aspiration, or if the gastric mucosa is not able to reabsorb it. The latter condition is seen in ectasies of high degree that are genuine or are caused by scar tissue. Gluzinski expresses the opinion that in cases of ectasy of the stomach of high degree a decrease or a disappearance of the chlorids in the urine is an argument in favor of a benign process combined with excessive secretion of hydrochloric acid, and that it does not indicate the presence of a neoplasm.

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[1] *Lehrbuch d. Pathologie des Stoffwechsels*, Berlin, 1893.

[2] *Berlin. klin. Wochenschr.*, 1887, No. 28.

[3] *Sitzungsprotokoll d. IV. Cong. d. poln. Naturforscher und Aerzte*, June 2, 1884, Posen.

[4] *Berlin. klin. Wochenschr.*, 1887, No. 41.

[5] *Ibid.*, 1887, No. 52.

Stroh[1] repeated some of these experiments in my clinic and was unable to corroborate the last statement. The absolute values for chlorin in the urine can probably not be utilized for differential diagnosis because the absorption of food is interfered with in every case of stenosis of the pylorus, and this alone may lead to a decreased excretion of chlorin. F. Müller,[2] indeed, found a considerable decrease in the excretion of chlorids in all the cases of carcinoma that he examined. Laudenheimer[3] analyzed the amount of chlorin introduced with the food and found that in the case of carcinoma no typical aberration from the normal proportion between excreted and ingested chlorin existed.

The Phosphates.—The excretion of phosphates has not been sufficiently investigated in diseases of the stomach, at least not to the extent of determining the phosphates ingested and the phosphates in the combined excretion of the urine and the feces. According to Robin,[4] the amount of phosphates excreted is increased in hyperchlorhydria. F. Müller[5] found an increased excretion of P_2O_5 in carcinoma—not, however, in all cases.

The Sulphates.—In pathologic cases the conjugate sulphates (ethereal sulphates) are of more interest than the preformed sulphates. The excretion of total sulphates runs parallel to the anabolism of albumin.

We know that the hydrochloric acid of the stomach is a bacterial poison, although not an absolute germicide; a decrease of the hydrochloric acid in the stomach will favor the development of bacteria. Von Noorden,[6] in opposition to Kast[7] and Wasbutzki,[8] who found the excretion of conjugate sulphates increased in hydrochloric acid deficiency, found an increase of the ethereal sulphates only five times in 11 cases in which the secretion of hydrochloric acid was almost *nil.* He performed 46 determinations in all. Only in a small proportion of cases was the excretion of ethereal sulphates greater than in healthy subjects, showing that the putrefaction of albumin in the intestine was not increased above normal; in fact, in the majority of his cases the values found were below the normal average; Boas[1] found the same in a case of stenosis of the duodenum in which there was permanent absence of hydrochloric acid. On the other hand, Mester[2] has shown that the ethereal sulphates may be very much increased if substances that are already in process of putrefaction enter a stomach that contains no hydrochloric acid. The absence of hydrochloric acid in the stomach *per se* exercises no influence on the ordinary putrefactive processes in the intestine.

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[1] *Inaug. Diss.*, Giessen, 1888.

[2] *Zeitschr. f. klin. Med.*, vol. xvi., p. 496.

[3] *Ibid.*, vol. xxi., p. 513.

[4] S. Lyon, *L'analyse du suc gastrique.*

[5] *Zeitschr. f. klin. Med.*, vol. xvi.

[6] *Hamburger Festschrift*, 1889.

[7] *Arch. f. exper. Pathol. u. Pharm.*, 1889, vol. xxvi., 133.

[8] *Loc. cit.*

[The suggestion of Simon, that indicanuria was to some extent dependent upon hypochlorhydria for its production, is now generally rejected.

Allen Jones[3] has shown that it is usually present in gastric ulcer hyperchlorhydria—that it is very often absent in cases where the secretion of hydrochloric acid is greatly reduced or absent. As indicanuria has been shown to depend upon the putrefaction of albuminous substances in the intestine, it is natural to question the stomach digestion for some contributing causative influence. When the intestinal digestion is deficient, recent work makes it wholly probable that the depression of gastric juice may favor putrefaction. However this may be, in the light of our present knowledge the question is of no practical importance.—Ed.]

Nitrogen.—It is self-evident that serious diseases of the stomach that lead to malnutrition also cause a loss of body proteid. The same is seen in healthy subjects that are in a state of subnutrition.

Von Noorden's[4] communications on general metabolism, or, better, on the katabolism of proteid in acute diseases of the stomach, are the only data we possess. Von Noorden reports a case of gastro-enteritis with vomiting and diarrhea in which the loss of nutritive material was greater than it would have been in simple inanition. At the same time other factors injured the body-albumin—probably toxins that were absorbed from the intestine. In chronic diseases of the stomach protoplasmic poisons may also be absorbed and lead to the decomposition of proteid. Von Noorden has performed metabolic examinations in gastric ulcer, in chronic catarrh of the stomach, in gastrectasy with hypersecretion, but failed to find any abnormal increase of proteid katabolism. Von Noorden, therefore, believes no other factor than malnutrition is active in the majority of cases of chronic stomach trouble. F. Müller,[5] on the other hand, has performed a number of exact determinations in cases of carcinoma, and has found that in the majority of instances the katabolism of the body-proteid was greater than would correspond to simple malnutrition. He showed that the amount of nitrogen excreted was always greater than the amount of nitrogen ingested, even if abundant food was given. Klemperer,[1] Gärtig, and von Noorden[2] later corroborated these discoveries of Müller, and showed that his experiments on the increased katabolism of proteid were correct.

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[1] *Deutsch. med. Wochenschr.*, 1891, No. 28.

[2] *Zeitschr. f. klin. Med.*, vol. xxiv.

[3] *New York Med. Jour.*, April 20, 1900.

[4] *Zeitschr. f. klin. Med.*, vol. xvii.

[5] *Ibid.*, vol. vi.

It was found, however, that the katabolism of proteid is not increased in every case of carcinoma. Müller, from the fact that small carcinomata (even if they are situated in some location where they do not interfere with the functions of the body) are still able to damage the nutrition of the whole organism, draws the conclusion that certain metabolic products are formed in these carcinomata that are toxic. No one has, however, so far succeeded in isolating or identifying these toxic products of cancer.

Ferments.—Normally, pepsin and rennet are found in the urine. No diagnostic significance attaches to the presence of pepsin. Leo,[3] Stadelmann,[4] and others have shown that in carcinoma of the stomach the pepsin excreted in the urine is reduced or absent; but such a reduction in the pepsin excretion has also been seen in other diseases. All the investigations published so far are insufficient to allow us to draw any diagnostic conclusions from the amount of pepsin excreted in the urine. The same applies to rennet. Even in the same individual the amount of this ferment excreted may fluctuate within wide boundaries (Boas).

Albumin.—Small quantities of albumin are found in the urine in the later stages of severe diseases of the stomach. The presence of albumin is of no pathognomonic significance. Von Noorden[5] found considerable quantities of albumin in the urine after violent attacks of gastric pain in ulcer and after severe gastric hemorrhages. Müller[6] has collected the histories of all the cancers of different organs that occurred in the Würzburg Clinic, and found that in 35 per cent. there was albuminuria of a transitory character. He found the same in 72 per cent. of the cases in the second medical clinic in Berlin.

Peptones and Albumoses.—Some investigators, for instance, von Noorden,[7] deny the occurrence of true peptone in the urine; others, again, claim to find it. The reason for this diversity of opinion is that the views in regard to the exact chemical identity of peptones and albumoses are so much at variance. I cannot discuss the purely physiologic question as to what constitutes true peptone. I refer for this question to von Noorden and to a dissertation by Robitschek.[8] From a clinical point of view it is important to know that in certain diseases proteid substances are found in the urine that do not give the nitric acid nor the acetic acid ferrocyanid test, but do give the biuret reaction. In the normal urine we know, on the other hand, that these proteid bodies are never seen. It is of subordinate importance whether or not they are to be considered more or less pure peptones.

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[1] *Berlin. klin. Wochenschr.*, 1889, No. 40, and *Annales de la Charité*, vol. xvi.

[2] Gärtig, *Inaug. Diss.*, Berlin, 1890.

[3] *VII. Cong. f. innere Med.*, 1888, p. 364.

[4] *Zeitschr. f. Biol.*, 1889, xxv., 208.

[5] *Lehrbuch der Pathologie des Stoffwechsels*, Berlin, 1893.

[6] *Zeitschr. f. klin. Med.*, vol. xvi.

[7] *Loc. cit.*, p. 256.

[8] *Zeitschr. f. klin. Med.*, vol. xxiv.

The excretion of this form of proteid is called enterogenous peptonuria by von Jaksch.[1] Meixner[2] found it in 12 cases of carcinoma of the stomach. Robitschek, on the other hand, found it only twice among 7 cases; he also found it in gastric ulcer. Pacanowski[3] employed Hofmeister's[4] method for demonstrating the presence of peptone in the urine, and found peptonuria in carcinoma of different organs, not of the stomach alone. No definite diagnostic conclusions can be drawn from the occurrence of peptonuria.

Acetone and Diacetic Acid.—Kaulich[5] was the first to call attention to the appearance of acetone in the urine in diseases of the stomach and intestine. Cantani[6] corroborated his discovery. Von Jaksch[7] has also performed some very thorough investigations in regard to the excretion of acetone and diacetic acid. Lorenz[8] has published some exhaustive work on the relationship between acetonuria and digestive disorders. According to these authors, acetonuria occurs regularly in digestive disorders of various kinds. Lorenz does not believe that diaceturia and acetonuria can be separated. In the greater number of severe digestive disturbances a combination of acetonuria and diaceturia is usually seen, or the excretion of these two substances may alternate. According to our present knowledge, the appearance of acetonuria and its congeners does not justify us in drawing any other conclusion than that proteid katabolism is increased. The same applies to the occasional appearance of indican and indigo-red in the urine.

[Acetonuria is coming to be regarded as the result of disturbance of the digestion or metabolism of the fats rather than of the proteids and carbohydrates. Waldvogel[9] administered to fasting persons a diet of pure proteids and carbohydrates, but in neither case did acetone appear in the urine. When, however, fats were fed to starving people, acetonuria developed. As this result did not follow the subcutaneous administration of fats, it is believed that this acetonuria was dependent upon some alterations set up in the gastro-intestinal tract. He also confirms the general belief that eating of carbohydrates in some measure inhibits the development of acetone. It is known that acetone may be chemically produced from fats, but attempts to obtain it from proteids have been unsuccessful, although Blumenthal and Neuberg[10] have obtained it from gelatin, which suggests that it is possible to obtain similar results from proteid substances.—Ed.]

THE BLOOD IN DISEASES OF THE STOMACH.

The blood has been examined in all directions in diseases of the stomach, but so far no results that are diagnostically valuable have been obtained. Many investigators hoped to be able to discover some points that would be of value in the differential diagnosis between malignant diseases of the stomach, carcinoma, etc., on the one hand, and benign disease on the other. This expectation has so far not been fulfilled. Strasser[1] has performed some very careful investigations in regard to the number of red blood-corpuscles, the quantity of albumin of the total blood and of the serum, the specific gravity of the blood, etc., and has found that all these factors are subnormal in carcinoma, and that the impoverishment of the blood in the above sense increases progressively with the development of the disease. In the cachectic stage the number of red blood-corpuscles, the amount of proteid, and the specific gravity of the blood were all considerably diminished, whereas in the beginning of these diseases these abnormalities were not so pronounced. Strasser also found an increase of the white blood-corpuscles in the majority of cases. His findings agree with other statements that were made before his work was published. He also showed that in carcinoma all forms of poikilocytes appeared, whereas in tuberculosis the form of the red blood-corpuscles was not changed. None of these differences, however, are characteristic, nor can they be utilized in making a differential diagnosis.

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[1] *Zeitschr. f. klin. Med.*, vol. vi., viii., and *Zeitschr. f. physiol. Chemie*, vol. vi.

[2] *Zeitschr. f. klin. Med.*, vol. viii.

[3] *Ibid.*, vol. ix., p. 428.

[4] *Zeitschr. f. physiol. Chemie*, vol. iv.

[5] *Prager Vierteljahrsschr.*, 1857, vol. xiv.

[6] Abstract from *Schmidt's Jahrb.*, 1865, 127, 167.

[7] *Zeitschr. f. klin. Med.*, vol. viii.

[8] *Ibid.*, vol. xix.

[9] *Centralbl. f. innere Med.*, July 15, 1899.

[10] *Deutsch. med. Wochenschr.*, vol. xxvii., p. 6.

Analogous changes have been found in other diseases of the stomach; even in the case of the same disease the reports of different authors are conflicting. Leichtenstern,[2] for instance, found a reduction of the hemoglobin in 3 cases of ulcer. Oppenheim,[3] on the other hand, did not succeed in demonstrating such a reduction in 12 cases, and Reinert[4] finally failed to find either a reduction of hemoglobin or a reduction in the number of red blood-corpuscles. Osterpey[5] is undoubtedly right when he says that neither normal nor abnormal blood-pictures are characteristic for ulcer of the stomach. The blood-picture, as has been shown in carcinoma, depends on two factors—the age of the ulcer and the general condition of the patient; thus it will be different if the ulcer is recent and the patient robust, than if the ulcer is an old one and the subject anemic as the result of violent gastric hemorrhages.

No clues for a differential diagnosis between diseases of the stomach can, therefore, be obtained from the examination of the blood, and the number of red and white blood-corpuscles and the percentage of hemoglobin give us no information whatever.

The determination of the specific gravity of the blood, according to Schmaltz,[6] can hardly be utilized for diagnostic purposes, even though different authors claim that in carcinoma of the stomach the specific gravity of the blood is considerably reduced. In cases of anemia the same reduction has been seen; the fact that in benign diseases of the stomach, in which general nutrition is not impaired, the specific gravity is normal cannot be utilized in the differential diagnosis. The reduction in the specific gravity runs parallel with the reduction in the general nutrition of the patient.

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[1] *Zeitschr. f. klin. Med.*, vol. xxiv.

[2] Leichtenstern, *Untersuchungen über den Hämoglobingehalt im gesunden und kranken Zustande*, Leipsic, 1878.

[3] *Deutsch. med. Wochenschr.*, 1889, Nos. 42–44.

[4] Reinert, *Die Zählung der rothen Blutkörperchen und deren Bedeutung für Diagnose und Therapie*, Leipsic, 1891.

[5] *Berlin. klin. Wochenschr.*, 1892, Nos. 12, 13.

[6] *Deutsch. Arch. f. klin. Med.*, vol. xvii., and *Deutsch. med. Wochenschr.*, 1891.

The Alkalescence of the Blood.—The investigations of Sticker and Hübner,[1] von Noorden,[2] and others have shown that normally the alkalescence of the blood is increased at the height of gastric digestion—that is, at the time when the secretion of hydrochloric acid is abundant. It was to be expected, therefore, that considerable differences in the alkalinity of the blood would be found in cases of hyperacidity or of anacidity of the stomach. Von Noorden[3] has investigated this subject and has found that the differences in the blood alkalinity are so small that they can be explained by technical errors. At the same time we believe that this is a prolific field for investigation, and that it will be worth while to continue investigations in this direction, particularly as so little has been done on this subject.

The number and the appearance of the white blood-corpuscles have been studied at the height of digestion, and some authors have attempted to utilize certain differences for diagnostic purposes. We know that at the height of the digestion of albuminous food there is a transitory leukocytosis. Pohl[4] was the first to investigate this so-called digestion leukocytosis, and to determine that it appears after the introduction of proteid food. It is dependent on the amount of albumin and albuminoid material ingested; the carbohydrates, fats, salts, water, and the non-proteid constituents of the food do not produce leukocytosis. Pohl's investigations have also shown that at the same time an increased emigration of lymph-cells occurs from the mucous lining of the intestine into the venous blood.

Schneyer[5] has studied this digestion leukocytosis in 30 cases of carcinoma and ulcer. He found that in all his cases of carcinoma (18) digestion leukocytosis was absent, whereas in ulcer cases, with the exception of 1 case, which was marantic, it was present. Hartung[6] repeated these experiments and arrived at the same conclusions. Digestion leukocytosis is even absent in cases of carcinoma if nuclein, which Horbaczewski[7] has given in the place of the corresponding amount of food, is administered, and we know that nuclein is capable of producing an artificial leukocytosis within a few hours. Schneyer attributes the absence of digestion leukocytosis in carcinoma to disturbances of the resorptive powers, of the peptonization of the albumin, and of the lymphatic apparatus. He believes that the absence of digestion leukocytosis does not necessarily indicate the presence of a carcinoma, but that, on the other hand, the presence of digestion leukocytosis certainly does militate against the diagnosis of carcinoma.

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[1] Sticker and Hübner, *Zeitschr. f. klin. Med.*, vol. xii., p. 136; also Sticker, "Verhandl. des allg. ärztl. Vereines zu Köln," *Deutsch. med. Wochenschr.*, 1888.

[2] Von Noorden, *Arch. f. exper. Pathol. u. Pharm.*, 1888, vol. xxii., p. 325.

[3] *Ibid.*, *Lehrbuch der Pathologie des Stoffwechsels*, 1893, p. 246.

[4] Pohl, "Ueber Resorption und Assimilation der Nährstoffe," *Arch. f. exper. Pathol. u. Pharm.*, vol. xxv.

[5] Schneyer, "Das Verhalten der Verdauungsleukocytose bei Ulcus rotúndum und Carcinoma ventriculi," *Internat. klin. Rundschau*, 1894, vol. viii., No. 39, und *Zeitschr. f. klin. Med.*, vol. xxvii., Nos. 5, 6.

[6] *Wien. klin. Wochenschr.*, 1895, Nos. 40, 41.

[7] *Allg. Wien. med. Zeit.*, 1892, No. 32.

These conclusions of Schneyer cannot, however, be adopted without reserve, for we know that there are patients with carcinoma that enjoy a good appetite and eat meat. This, of course, applies only to the earlier stages, where the motor powers of the stomach are still intact, so that the intestine vicariously performs the peptic functions of the stomach. It has also been seen that many cases of carcinoma regain their appetite and their strength after gastro-enterostomy, and we can hardly expect that in cases of this character digestion leukocytosis will be absent, provided they eat albuminous food. The cause for the absence of digestion leukocytosis is the fact that carcinomatous cases usually dislike albuminous food because they cannot absorb it. This is more or less theoretic, however, and we must await the results of exact experimentation in this direction.

It is natural, of course, that digestion leukocytosis should be absent in later stages of carcinoma when the appetite is greatly reduced and there is a pronounced dislike for meat. For this reason the symptom we are discussing is probably without value in differential diagnosis, as the carcinoma itself is not responsible for the absence of digestion leukocytosis.

The expectation of finding valuable clues for the differential diagnosis of diseases of the stomach in the blood has not been fulfilled, and, however interesting some of the results obtained may be, they are as yet devoid of practical importance.

[The more recent investigations of Douglas,[1] Osler, and McCrae[2] all go to show that no reliance can be placed upon digestion leukocytosis as an aid to the diagnosis of cancer of the stomach. It seems that only about 54 per cent. of cases gave positive reaction in this regard. It has been shown by A. Japha[3] that in young children, whether sick or well, digestion leukocytosis is not a regular phenomenon, and in them, therefore, lacks diagnostic value.

Krokewicz[4] found that of 17 cases of carcinoma of the stomach, in 13 no digestion leukocytosis was observed; in 4 cases it was present, and in 2 of these it was constant.

Roman Rencki[5] says that he found the absence of digestion leukocytosis in cases of insufficiency or incompetency of the pylorus. Finding the absence of digestion leukocytosis in cases of stenosis of the pylorus, he argues that the same result occurs when the pylorus is incompetent, for the reason that there is a more or less continuous passage into the duodenum of small amounts of food, so that there is no special stimulation of leukocytosis at any one time.

In a painstaking study of the subject of digestion leukocytosis in carcinoma Rencki finds that it is by no means constantly absent. It may be delayed, and sometimes occurs as late as six to fourteen hours after the ingestion of food. The absence of this leukocytosis does not appear to depend upon the extent of degeneration of the gastric mucosa, as in one case in which there was wide-spread involvement of the gastric mucous membrane, there was a marked digestion leukocytosis. Study of a large number of cases of all kinds leads Rencki to conclude that the absence of digestion leukocytosis is dependent upon the stenosis of the pylorus. He finds that wherever this condition exists, absence of leukocytosis is to be expected. In some instances in which leukocytosis was absent he made the interesting experiment of introducing an ample nutrient enema, and found that a digestion leukocytosis resulted.

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[1] *Brit. Med. Jour.*, March 16, 1901.

[2] *Cancer of the Stomach*, 1900, p. 119.

[3] *Jahrbuch f. Kinderheilkunde.*

[4] *Arch. f. d. Verdauungskrankheiten*, vol. vi., No. 1.

[5] *Ibid.*, vol. vii., Nos. 4, 5.

The same investigator made a very careful study of the red blood-cells in cases of both carcinoma and gastric ulcer, and found that there resulted a considerable deterioration of blood in both diseases, but the changes were not characteristic; there was discovered merely a secondary anemia which may follow from any one of various affections that seriously interfere with nutrition. In case of ulcer the depreciation in blood was sometimes secondary to vomiting or lack of food, sometimes to frequent hemorrhages.—Ed.]

THE TREATMENT OF DISEASES OF THE STOMACH IN GENERAL.

Introductory Remarks.—In order to institute a rational therapeusis of diseases of the stomach a precise diagnosis must be made. This presupposes an exact knowledge of the different perversions of function, and not only the determination of the pathologico-anatomic changes. We do not, as a rule, treat anatomic changes, but we attempt to remedy the disturbances of function, and, by removing the latter, create favorable conditions for the cure of the former; even in those cases where a cure is impossible symptomatic treatment may do much good. For instance, ectasy of the stomach may be relieved by a carefully selected diet and by methodic lavage. Fermentation in cases of hypersecretion may be relieved by the administration of antifermentative remedies, or, again, gastro-enterostomy may be performed in cases of stenosis of the pylorus.

If we except the sensory disturbances of the stomach, the perversions of the organ may be three-fold—either the secretion of gastric juice, the motility, or the absorptive powers may be perverted.

The gastric secretion has two functions—first, to dissolve the proteid substances; second, to inhibit putrefactive and fermentative processes. The hydrochloric acid of the stomach is capable of performing this latter function only within certain boundaries, and, above all, only so long as the motor power of the stomach is intact.

The motor function is intended to mix the food thoroughly with the gastric juice, to break the food up, and, finally, to propel it into the intestine. The absorptive function of the stomach is less important than the other two functions enumerated, and is not so independent.

Much importance is usually attached to disturbances of gastric secretion; in practice, however, we find that disturbances of the motor function are more important than disturbances of the secretory function. As long as the motor power of the stomach is good, a simple reduction of the secretory function can be borne without detriment to the organism. This is due to the fact than the intestinal secretion, particularly the pancreatic juice, can vicariously assume the peptic function of the stomach, provided the motor power of the organ is good; if the motor power, on the other hand, is disturbed, the ingesta remain in the stomach for an abnormal length of time, so that they do not enter the intestine until after fermentation and decomposition have occurred. These facts explain why certain cases of benign stenosis of the pylorus are accompanied by severe disturbances of the general nutrition, whereas cases of greatly decreased secretory power may enjoy comparatively good health so long as the motor power of the stomach is good.

Perversions of the secretory and motor functions of the stomach may occur in two directions: Both functions may be perverted in the same sense—that is, both may be increased or decreased; frequently, however, the perversions occur in an opposite sense; finally, there are cases where only the one or the other function is perverted. At present we know comparatively little about the disturbances of absorption. It is probable that a perversion of this function rarely occurs alone, but usually accompanies disturbances of secretion and of the motor power.

We have many means of counteracting these different functional disorders. The remedy should be selected according to the individuality of each case. This is particularly true in diseases of the stomach.

In the following we will discuss the means at our disposal in general, and also, particularly, in regard to the different forms of functional disorders of the stomach.

We have two practical methods for curing or alleviating disorders of the stomach—first, dietetic measures; secondly, measures that are intended to cure or to relieve. The latter can be subdivided into—(*a*) medicamentous measures proper; (*b*) physical methods and remedies.

Following a discussion of all these remedies we will mention briefly the surgical treatment of diseases of the stomach. I will not, however, discuss the prophylaxis of stomach-diseases in this place, because we will have to recur to this subject in discussing the etiology of the different diseases of the stomach. General dietetic measures are as important for prophylaxis as for the cure of diseases of the stomach, and for this reason we will mention briefly some of them in discussing the treatment.

Prophylaxis of diseases of the stomach should begin in childhood. The stomach, like any other organ, should be educated from childhood up. Prophylactic measures in each individual should extend to the whole digestive apparatus, the care of the teeth, the manner of eating, the digestibility of different articles of food, the quality and quantity of the food and drink, the frequency of meals, and numerous other points. We will refer to some of these factors that are important for the pathology of diseases of the stomach in the subsequent course of this discussion.

THE DIET.

The Quality of the Diet According to the Different Diseases of the Stomach.—In order to maintain health and to prevent diseases of the stomach, certain dietetic rules should be observed. After the stomach is diseased the selection of a suitable diet is particularly important. In the treatment of diseases of the stomach the diet and the selection of food are as important, or possibly more important, than treatment by drugs. In this place we will limit ourselves to discussing a few general dietetic principles. The diet will be different in the different diseases, and will, in addition, have to be adapted to each individual patient; for instance, a case of carcinoma with good motor power will demand a different diet than a case of carcinoma with ectasy of high degree, atony, and lactic-acid fermentation.

It has been frequently stated that the diet of a case of stomach-disease should be easily digestible. While it is an easy matter to determine what is easily digestible in a healthy subject, this is not so simple in disease of the stomach. In one form of stomach-disease a certain article of diet may be easily digestible ; in another form it will not be ; an article of diet may be digested easily and still not be well borne by the patient; the two terms are by no means synonymous, so that we may say that an article of diet that is digested without difficulty may, at the same time, not be well borne ; and, inversely, that an article of diet is not digestible at all and may still be well borne by the patient. In pathologic cases, therefore, the term "easily digestible" is variable. In general, we may say that an article of diet is easily digestible if it makes small demands on the secretory and motor functions of the stomach—that is, if it is readily absorbed and causes no subjective disturbances. This definition, at the same time, applies to articles of food that are well borne. A scale of digestibility of different articles of food may be arranged according to the length of time that they remain in the stomach. Leube,[1] and after him more extensively Penzoldt,[2] has done this. A scale of this kind cannot, however, be applied to pathologic cases, and each disease will have to be judged separately. A certain class of food may be easily digestible for a case of carcinoma of the stomach and not so for a case of ulcer. The length of time that the article of food remains in a healthy stomach may in general be regarded as an indicator of its digestibility, for it indicates not only the work of the motor function, but also, to an extent, the work of the secretory function of the stomach, as both these functions usually act together in health. In pathologic cases this is different, for here the one or the other of these functions, or both, may be perverted, sometimes in opposite directions. In addition, we have seen that gastric digestion is occasionally vicariously assumed by the intestine ; here we find that the food does not remain in the stomach a long time, notwithstanding the fact that the function of the stomach is very much reduced. We see, for instance, in rare cases that free hydrochloric acid is completely absent, but that the food does not remain in the stomach for an abnormal length of time. If we wash out the stomach four or five hours after a test-meal, we will find the organ empty; if we wash it out some time before this, we will find that the peptic power of the stomach is very much reduced. Here, then, the food does not remain in the stomach for an abnormal length of time, and still gastric digestion is certainly subnormal. For all these reasons a scale that is arranged according to the length of time that food remains in the normal stomach cannot be applied to a great number of pathologic cases. If the vicarious action of the intestine completely compensates the deficient peptic powers of the stomach, we can state that the digestibility of certain articles of food is good or that there is good intestinal digestion.

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[1] *Zeitschr. f. klin. Med.*, vol. vi.

[2] *Deutsch. Arch. f. klin. Med.*, vol. li., liii.

From the point of view of pathologic anatomy the universal employment of such terms as anacidity, subacidity, hyperacidity, hypersecretion, atony, hypatony, etc., may be condemned; clinically, however, we cannot get along without them, and it is a matter of great importance in prescribing the diet in each individual case to know whether there is subacidity or hyperacidity, atony or hypatony, etc.

At the time when Leube performed his fundamental experiments on the digestibility of different articles of food our knowledge of the different perversions of function which we have discussed above was very deficient. Leube's experiments, therefore, were carried on in the right direction when he attempted to determine what articles of food were easily digestible by determining the length of time required by a diseased stomach to digest each article that he investigated. In addition, he determined the subjective disturbances complained of during the digestion of different articles of food, as well as the influence that certain articles of diet exercised on the general disease of the stomach. For his experiments Leube selected cases in which the stomach function was very much impaired. On the assumption that a healthy stomach can digest and get rid of a moderate amount of food in from six to seven hours, he concluded that any article of food that was removed from the stomach of a patient within this time-limit was easily digestible, whereas if it was not removed within this time it was not easily digestible. Leube[1] constructed a scale of foods according to their digestibility that is valid to-day and forms the basis of our dietary regulations in gastric ulcer. In other diseases of the stomach it does not apply so well. I feel warranted in inserting Leube's diet-scale in this place (see table):

LEUBE'S DIET-SCALE.

DIET I.—If the digestion is very much reduced, the following articles of food are most easily digestible: bouillon, meat solutions, milk, raw or soft-boiled or poached eggs.

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[1] *Zeitschr. f. klin. Med.*, vol. vi.

Diet II.—Less digestible than diet I. are the following articles of food: boiled calves' brain, boiled thymus, boiled chicken, and pigeon. These different kinds of meat are enumerated in the order of their digestibility. Other articles of food that are permissible are gruels, and in the evening milk mushes made with tapioca and white of egg. The majority of patients can assimilate boiled calves' feet in addition to the articles of meat mentioned.

Diet III.—If diet II. can be digested, diet III. follows. The increase consists in adding cooked or raw beef to the above diet-list. Leube mentions the following method of preparing beefsteak, and claims that beef cooked in this way is very easily digestible. The meat should be allowed to lie for some time and scraped with a dull spoon; in this way a meat-pulp is obtained consisting only of the delicate parts of the muscle, and containing none of the tough, hard, and sinewy portions. These meat-scrapings are roasted in fresh butter. Raw ham is also permissible in this stage.

In addition to meat a little mashed potato may be given, some white bread that is not too fresh, and possibly small quantities of coffee or tea with milk.

Diet IV.—Roast chicken, roast pigeon, venison, partridge, roast-beef, medium to raw (particularly cold), veal (from the leg), pickerel, boiled shad (trout, even young ones, are very hard to digest), macaroni, bouillon with rice. Small quantities of wine to be taken one to two hours before eating; gravies are contraindicated. Young and finely chopped spinach is the best vegetable; other vegetables, as asparagus, may be tried, although Leube considers this a risky procedure. The patients are allowed to take a more liberal diet after this fourth diet, but the increase should be very gradual. They should refrain from eating vegetables, salads, and preserves and fruits for a long time. The first of these articles that they may eat is baked apple.

Penzoldt[1] has recently repeated these experiments of Leube's, but not, as Leube, in cases of stomach-disease, but in subjects with normal stomachs. He also constructed his scale from the length of time that the different articles of food remained in the stomach. His experiments are particularly valuable because they were performed in healthy subjects—that is, under equal conditions. Their value is still more enhanced by the fact that the quantity of food ingested, the hydrochloric acid secretion, and other factors were considered at the same time; and while these results cannot be applied directly to cases of stomach-disease, they still furnish some very valuable clues. I feel justified, therefore, in reproducing in this place Penzoldt's scale of the most important articles of food and drink, giving the amounts of each article. I think that a scale of this kind should form the basis of all our dietary regulations in pathologic cases (see table):

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[1] *Deutsch. Arch. f. klin. Med.*, vol. li., liii.

The following articles of food left the stomach within—

One to two hours.

100-200 gm.	pure water.
220 gm.	carbonated water.
200 gm.	tea, alone.
200 gm.	coffee, alone.
200 gm.	cocoa, alone.
200 gm.	beer.
200 gm.	light wines.
100–200 gm.	boiled milk (A. 3.5, F. 3.5, C. 5[1]).
200 gm.	meat-broth, alone.
100 gm.	eggs, soft.

Two to three hours.

200 gm.	coffee with cream.
200 gm.	cocoa with milk.
200 gm.	Malaga wine.
200 gm.	"Ofen" wine.
300–500 gm.	water.
300–500 gm.	beer.
300–500 gm.	boiled milk.
100 gm.	eggs, raw and scrambled, hard boiled or as omelet (A. 12, F. 12).
100 gm.	beef-sausage, raw.
250 gm.	calves' brains, boiled.
250 gm.	calves' thymus, boiled.
72 gm.	oysters, raw.
200 gm.	carp, boiled.
200 gm.	pike, boiled (A. 18, F. 0.5).
200 gm.	shellfish, boiled (A. 17, F. 0.05).
200 gm.	cod, boiled (A. 80, F. 1).
150 gm.	cauliflower, boiled (A. 2, C. 4).
150 gm.	cauliflower as salad.
150 gm.	asparagus, boiled (A. 2, C. 2).
150 gm.	potatoes boiled in salt water (A. 2, C. 20).
150 gm.	mashed potatoes.
150 gm.	stewed cherries.
150 gm.	raw cherries.
70 gm.	white bread, old or fresh, dry or with tea (A. 7, C. 52).
70 gm.	pretzels.
70 gm.	Zwieback, fresh or stale, dry, or with tea.
50 gm.	Albert biscuits.

Three to four hours.

230 gm.	young chicken, boiled (A. 20, F. 4).
230 gm.	partridge, broiled.
220–260 gm.	pigeon, boiled.
195 gm.	pigeon, fried.
250 gm.	beef, raw, boiled, lean (A. 21, F. 1.5).
250 gm.	calves' feet, boiled.
160 gm.	ham, boiled (A. 24, F. 36).
160 gm.	ham, raw.
100 gm.	veal, warm and cold, lean (A. 20, F. 1.5).
100 gm.	beefsteak, broiled, cold or warm.
100 gm.	beefsteak, raw, scraped.
100 gm.	tenderloin.
200 gm.	Rhine salmon, boiled (A. 16, F. 28).
72 gm.	caviar, salted (A. 31, F. 16).
200 gm.	sardines in vinegar, kippered herring.
150 gm.	blackbread (A. 6, F. 0.5, C. 50).
150 gm.	barley bread.
150 gm.	wheat bread.
100–150 gm.	Albert biscuits.
150 gm.	potato as vegetable.
150 gm.	rice, boiled (A. 3, C. 76).
150 gm.	Kohlrabi, boiled (A. 3, C. 8).
150 gm.	carrots, boiled (A. 1, C. 9).
150 gm.	spinach, boiled.
150 gm.	cucumber salad.
150 gm.	radishes, raw.
150 gm.	apples.

Four to five hours.

210 gm.	pigeon, broiled.
250 gm.	fillet of beef, broiled.
250 gm.	beefsteak, broiled.
250 gm.	beef-tongue, smoked (A. 24, F. 31)
100 gm.	smoked beef in slices (A. 27, F. 15).
250 gm.	hare, broiled.
240 gm.	partridge, broiled.
250 gm.	goose, broiled (A. 16).
280 gm.	duck, broiled.
200 gm.	herring, salted.
150 gm.	lentils, mashed (purée) (A. 25, C. 54).
200 gm.	peas, as purée (E. 23, K. 52).
150 gm.	string-beans, boiled (A. 3, C. 6).

Guided by the length of time that different articles of food remain in the stomach, and considering at the same time the method of preparation, the quality, and the other properties of different articles of food and drink, Penzoldt has also formulated four diet-lists. They correspond essentially with Leube's diet-lists, but have the advantage of giving the exact amount of each article of food. They are also more complete (see tables).

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[1] A. indicates the percentage of albumin, F. of fat, and C. of carbohydrates.

Diet I. (About Ten Days).

Food or drink.	Largest quantity to be taken at any time.	Method of preparation.	Special requirements.	How to be eaten.
Meat-broth.	250 gm. ($1\frac{1}{4}$ liter).	From beef.	Without fat, or not salted.	Slowly.
Cow's milk.	250 gm. ($\frac{1}{4}$ liter).	Well boiled or sterilized (Soxhlet apparatus).	Entire milk (or $\frac{1}{3}$ lime-water, $\frac{2}{3}$ milk).	If desired, with a little tea.
Eggs.	1 or 2	Very soft, just heated or raw.	Fresh.	If taken raw, should be stirred into the warm, not boiling, meat-broth.
Meat solution (Leube-Rosenthal).	30–40 gm.	. . .	Should have only a slight meat-broth odor.	In teaspoonful doses, stirred in meat-broth.
Cakes (Albert biscuits).	6	. . .	Without sugar.	Not softened, but should be thoroughly masticated and insalivated.
Water.	$\frac{1}{8}$ liter.	. . .	Ordinary water or natural carbonated water with a small percentage of carbonic acid gas (Seltzer).	Not too cold.

Diet II. (About Ten Days).

Food or drink.	Largest quantity to be taken at one time.	Method of preparation.	Special requirements.	How to be eaten.
Calves' brain.	100 gm.	Boiled.	Freed from all membranes.	Best taken in meat-broth.
Thymus from the calf.	100 gm.	Boiled.	As above, should be carefully enucleated from its capsule.	Best taken in meat-broth.
Pigeons.	1	Boiled.	Only if young, without skin, tendons, and the like.	Best taken in meat-broth.
Chicken.	1 as large as a pigeon.	Boiled.	As above (no fattened chicken).	Best taken in meat-broth.
Raw beef.	100 gm.	Chopped fine or scraped with a little salt.	From the tenderloin.	To be eaten together with cakes.
Raw beef-sausage.	100 gm.	Without any additions.	Smoked a little.	To be eaten together with cakes.
Tapioca.	30 gm.	Boiled with milk to make gruel.		

DIET III. (ABOUT EIGHT DAYS).

Food or drink.	Largest quantity to be taken at one time.	Method of preparation.	Special requirements.	How to be eaten.
Pigeon.	1	Broiled, with fresh butter.	Only young birds, skin, etc.	Without gravy.
Chicken.	1	Broiled, with fresh butter.	Only young birds, skin, etc.	Without gravy.
Beefsteak.	100 gm.	With fresh butter, half rare (English).	From the tenderloin, well beaten.	Without gravy.
Ham.	100 gm.	Raw, scraped fine.	Smoked a little, without the bone.	With white bread.
Milk bread or Zwieback or "Freiberger Pretzels."	50 gm.	Crisp, baked.	Stale (so-called rolls, etc.)	To be carefully masticated and well insalivated.
Potatoes.	50 gm.	(*a*) Mashed; (*b*) boiled in salt water and mashed.	The potatoes should be mealy and crumble on crushing.	
Cauliflower.	50 gm.	As a vegetable, boiled in salt water.	Use only the "flowers."	

These diet-lists, consisting of four different dietaries, are intended for the prophylactic treatment of certain diseases of the stomach (so-called ulcer cures of Leube). Their object is to spare the stomach in the beginning, and later to educate it up to increased activity by beginning with a very simple diet and gradually progressing to a more abundant and a more strengthening one.

It is evident that dietaries of this kind cannot be administered to all cases of stomach-disease in the same manner. The secretion of gastric juice, the motility, and the sensibility of the organ must be studied in every case. We will recur to a special description of the diet in the different diseases when discussing the diseases of the stomach. In this place we wish simply to give some general rules that apply to the principal types of stomach-disorders.

We need not mention the treatment of acute diseases of the stomach, for here the chief indication is to spare the organ as much as possible. We can limit our discussion to the question of diet in chronic diseases of the stomach. If we consider the secretion of gastric juice to begin with, we have, on the one side, hyperacidity and hypersecretion; on the other, subacidity and anacidity.

In the former group the stomach reacts to the introduction of food with more or less pronounced symptoms of secretory irritation—that is, by increased secretion of gastric juice. This symptom is frequently complicated by other symptoms of irritation in the sensory or even in the motor sphere.

In the second group this function is reduced and the secretion of gastric juice is more or less impaired. The motility at the same

DIET IV. (ABOUT EIGHT TO FOURTEEN DAYS).

Food or drink.	Largest quantity to be taken at one time.	Method of preparation.	Special requirements.	How to be eaten.
Venison.	100 gm.	Roasted.	From the back; should hang for a time, but not be "high."	
Partridge.	1	Roasted, without bacon.	Young birds, without skin, tendon, legs, etc.; should hang for a time.	
Roast-beef.	100 gm.	Medium to rare.	From good, fatted cattle; beaten.	Warm or cold.
Fillet of beef.	100 gm.	Medium to rare.	From good, fatted cattle; beaten.	Warm or cold.
Veal.		Roasted.	Back or leg.	Warm or cold.
Pike, Shad, Carp, Trout,	100 gm.	Boiled in salt water without any additions.	All fish-bones should be carefully removed.	In the fish gravy.
Caviare.	50 gm.	Raw.	Slightly salt, Russian caviar.	
Rice.	50 gm.	Mashed, pressed through a sieve.	Soft boiled rice.	
Asparagus.	50 gm.	Boiled.	Soft without any of the hard parts.	With a little melted butter.
Scrambled eggs.	2	With a little fresh butter and salt.		
Omelette (Souffle).	2 eggs.	With about 20 gm. of sugar.	Must have risen well.	To be eaten at once.
Fruit-sauce.	50 gm.	From fresh, boiled fruit, to be strained through a sieve.	Free from all kernels and peel.	
Red wine.	100 gm.	Light, pure Bordeaux.	Or some corresponding kind of red wine.	Slightly warmed.

time may be well preserved, and sensory disturbances are not frequently seen. The appetite in these cases is usually more or less reduced.

Cases in which the motility is increased are not important for the subject under discussion; those cases, however, must be considered a particularly important group in which the motility is more or less reduced—as, for instance, ectasy and atony. The character of the secretory perversion is, at the same time, an important point. Two forms can be distinguished: on the one hand, cases of gastrectasy and atony in which the secretion of gastric juice is normal or increased; on the other hand, cases in which the secretion is more or less reduced.

Stenoses of the pylorus from carcinoma and old cases of stomach-catarrh with atony belong to the latter group; benign stenoses of the pylorus, particularly as a result of cicatrices from ulcer, distortions of the region of the pylorus, and hypersecretion belong to the former group. Both forms are characterized by a great reduction in the motor power of the stomach.

These three groups could easily be subdivided still further. The quality and the quantity of the food and drink administered are, of course, different in each category. An article of diet that may readily be digestible in hyperacidity is not digestible at all in an acidity or subacidity.

Honigmann,[1] in a dissertation on the general dietetics of diseases of the stomach, has called the first group hyperacid or irritative forms. Here an increased secretion of gastric juice, and, at the same time, an increased sensibility of the organ, must be considered in prescribing. If there is simple hyperacidity without ulceration, a diet should be chosen that stimulates hydrochloric acid secretion as little as possible and at the same time combines as much of the hydrochloric acid as possible. In ulcer, which, as we know, is usually complicated by hyperacidity, a second factor must be considered—namely, the mechanical and chemical irritation that the diet may exercise.

I have already called attention to the fact that after an abundant test-meal the absolute acidity of the stomach is usually greater than after a small test-breakfast, but that after the latter meal it is relatively greater as compared to the amount of food ingested. It is important, above all, to know that the quantity of hydrochloric acid in excess, which really constituted the degree of hyperacidity, is occasionally the same, or even less, after a test-meal than after a test-breakfast. The irritation that the latter exercises cannot, therefore, be considered less than the irritation by an abundant test-meal. The hydrochloric acid that is overabundant in cases of hyperacidity is more completely utilized if a meal is administered containing much albumin than if a small meal is administered containing very little albumin. In simple cases of hyperacidity, therefore, an abundant albuminous diet should be given, because the proteid substances combine with the excessive hydrochloric acid.

[For years it has been theoretically held that albumins were most suitable for cases of hyperchlorhydria, and experience has sometimes confirmed this theory, but this is far from being always true, for not a few cases of hyperchlorhydria do better upon a diet of milk and bread than upon food of a strictly proteid character. The reason for this seems to be explained by Pawlow's work. Pawlow finds that the secretion of hydrochloric acid and the secretion of pepsinogen are by no means parallel, but this was already understood. He further shows that after eating bread the gastric juice shows the greatest amount of pepsin; after eating meat it is lower, and after milk, lowest of all. The acidity, on the other hand, is decidedly higher after the ingestion of meat and lowest after bread. It would, therefore, appear that the theory formerly held that albumin was most suitable in cases of hyperacidity, because of its ready combination with the acid, does not apply in those cases of excessive secretion in which the acid remains free in large amount after the combination with albumin has been satisfied. In other words, it cannot be laid down as a law that the albuminous foods are most suitable for all cases of hyperacidity, a fact which experience has long since shown. It is true that milk may have to be administered at frequent intervals, and it may be used between the meals at which albumins are taken. This latter plan is often very successful.

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[1] *Zeitschr. f. Krankenpflege*, 1894, Nos. 8, 9.

Arthur Meyer, of Berlin,[1] has experimented to determine whether more hydrochloric acid is secreted with egg-albumen than with amylaceous foods, and concludes, substantiating the results of others, that after meals of albumin more acid is found than after carbohydrates, and that free acid appears sooner with carbohydrates than with albumin. On this he bases the therapeutic indications that when distress is caused from free acid, it is best to administer albumins, but when the discomfort arises from the degree of the total acidity, amylaceous food should be advised.—Ed.]

The diet in cases of simple hyperacidity and in cases of hypersecretion that are not complicated by atony should consist of coarse albuminous substances given in the form of solid red meats. In addition, the meals should be abundant, but not given at too frequent intervals.

In ulcer the diet should be different; here all mechanical or chemical irritation should be avoided, so that finely distributed proteid substances or solutions of albumin are indicated. In all cases of simple hyperacidity or of hyperacidity complicated with ulcer, or of hypersecretion, amylaceous food is not well tolerated. This is particularly the case in hypersecretion; more so than in hyperacidity. In both cases we must remember that the hyperacidity of the stomach-contents hinders the digestion of carbohydrates. This inhibition of carbohydrate digestion is greater in hypersecretion than in hyperacidity. For this reason less carbohydrates should be given in the former condition than in the latter. In both conditions carbohydrates should be given in a form that is readily absorbed. Solutions of dextrose are particularly good, as they are readily absorbed and cause less secretion of hydrochloric acid than preparations of amylum.[2] They should be administered in moderation, and if there is only a slight degree of atony, they are altogether contraindicated because they favor fermentation.

The preparation of the food is also important. By the preliminary preparation of the food we give the diet a form and a consistence that aid its absorption and digestion by the gastric juice; at the same time we make the food more palatable. I refer in this respect to the diet-lists of Leube and Penzoldt, from which the great importance of the preparation of the food can be learned. It will be seen that the same article of diet may be easy or hard to digest according to their method of preparation. In hyperacidity the physician should know whether he wishes carefully to avoid irritation of the stomach or not, and he should choose his diet-list accordingly. Strong spices should, of course, always be avoided in these conditions, as they constitute a severe irritant for the stomach.

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[1] *Boas' Arch.*, vol. vi., pt. iii.

[2] S. Strauss, *Zeitschr. f. klin. Med.*, vol. xxix.

The mode of preparation is even more important in the case of vegetables than of meats. Here, too, we refer to the above diet-lists. We will refer to the different details later on.

In the second group the question of diet is simpler, because the appetite is reduced and the secretory functions of the stomach are impaired and at the same time the food given can be digested, provided the motor power of the stomach is intact. Here the decreased secretion of gastric juice must be particularly considered, and, at the same time, the food should not be too bulky in order not to tax the motor powers of the stomach, that are expected to compensate the secretory deficiency too much. Because of these considerations, it is best to give frequent small meals consisting of very tender meat that is finely chopped. In addition the patients may eat amylacea, tender vegetables, chopped spinach, cauliflower, asparagus, or mashed potatoes. The patients should also receive fat, preferably in the form of butter, as this form of food is most nourishing; at the same time an excess of fat should be avoided, and its administration stopped as soon as signs of irritation appear in the stomach or intestine.

The question of diet is most difficult in the third group—namely, in atony of high degree and ectasy of the stomach. Some of these cases cannot be treated by dietetic measures at all, because the only hope of improvement is surgical. I refer, for instance, to stenosis of the pylorus of high degree. In some of the cases methodic lavage may be indicated.

The diet will depend greatly on the secretion of gastric juice. If the secretion of gastric juice is chronically continuous, a nourishing meat-diet should be given and amylaceous food should be avoided. The ingestion of food should be reduced as in any case of atony; if necessary, fluid should be given by the rectum.

Still more difficult to treat are cases of atony in which the secretion of gastric juice is reduced and in which, consequently, albumin cannot be digested in the stomach; as the propulsion of the ingesta from the stomach into the intestine is interfered with in these cases, the intestine cannot possibly act vicariously, so that with the deficiency of hydrochloric acid, which prevents gastric digestion, nutrition becomes very much impaired. In cases of this kind albuminous food-products in solution, peptones, and albumoses may be tried; it seems that these articles are not suitable, however, for prolonged use. Other articles of food that may be tried are carbohydrates that are readily absorbed, and vegetables that contain much starch. Recourse may finally have to be had to nutritive enemata.

We have attempted to show in the preceding pages that the diet must be carefully selected in each case to fit the perversion of function existing. There is another factor, however, of equal importance that must be considered—namely, the appetite and the sensation of hunger that the patient experiences. In ordinary life and in healthy subjects the sensations play an important rôle, and it is particularly important that they should be considered in treating diseases of the stomach.

In a normal subject the demand of the organism for nourishment is manifested by the sensation of hunger—by the appetite. In the case of a person, however, who has been pampered and spoiled from childhood, or who has been accustomed to a monotonous diet, or, finally, who is suffering from some disease of the stomach, this regulating function is impaired.

The stomach, like every other organ, can be accustomed to perform a certain amount of work from childhood; if the diet is carefully and correctly selected, the resisting powers of the stomach are increased. If, on the other hand, the diet is too uniform, too monotonous, and not carefully selected, the stomach becomes pampered and less resistant to disease-producing agencies.

It is possible to educate the stomach to increased functional powers just as we can educate the heart and the lungs by a well-graduated system of exercise. Unfortunately, this training of the stomach is generally neglected. Our children, particularly in cities, are often spoiled, and the food they eat is administered more to suit the tastes or moods of the child than to nourish it, so that the stomach in these young persons is usually pampered and not capable of resisting disease. The result of all this is that in a subject of this kind the appetite and the sensation of hunger do not, in reality, reveal a true picture of the demands of the organism for nourishment.

Von Noorden[1] called attention to the fact that in healthy subjects the demand for food is reflected in the stomach. Under normal circumstances this picture is correct, and a healthy adult can maintain a uniform weight, barring slight fluctuations, for a period of years; in other words, healthy persons will eat just about as much as they can assimilate and use up. We do not know the path by which the cells of the body communicate the demand for nourishment to the higher centers; we may assume, however, that this path leads through the stomach.

As we have said, these sensations are a true picture of the demand of the organism for nourishment in normal subjects. In the case of persons who have been pampered and spoiled from youth upward or who are sufferers from some disease of the stomach this picture is no longer a true index. Here we may find that the relationship between the appetite, the sensation of hunger, on the one hand, and the individual demands for calories, on the other, is disturbed.

We do not know the way in which we become conscious of this demand for food; undoubtedly the central nervous organs are concerned in the process. A series of experiments have been performed in the expectation of elucidating this problem. For instance, rectal alimentation has been tried and the appetite and the demand for food experienced by the patient determined. It was found that the introduction of food into the rectum was capable of stopping the feeling of hunger to a certain degree, but the appetite could not be appeased in this way; even the introduction of abundant or overabundant food by the rectum could not produce this effect. The only way in which the appetite apparently could be appeased was by introducing food into the stomach.

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[1] *Berlin. Klinik*, 1893, No. 55.

In a healthy subject, therefore, the appetite and the feeling of hunger are an exact measure of the demands for calories. In diseases of the stomach, however, the appetite and the sensation of hunger no longer represent a true picture of these demands. We may say axiomatically that in the majority of diseases of the stomach, although not in all, the appetite is not so large as the demand of the cells for fuel. In the majority of diseases of the stomach we find the appetite reduced; in other words, smaller than the caloric demand. In other cases, which are rare, we see the proportion between the appetite and the demand for calories perverted in the opposite sense, so that the appetite is abnormally large and falsely simulates a great demand for calories.

In many diseases of the stomach there is great danger that the patient will take too little food because his appetite is diminished. As a result, patients of this kind are in a stage of chronic malnutrition. One of the chief duties of the physician is to administer so much food to the patient that the demand for calories is covered, provided, of course, that the forced introduction of food is not detrimental to the disease process itself. In order to do this intelligently the individual tastes of the patient should be carefully considered and ministered to.

In acute diseases of the stomach there is rarely any danger of inanition. In chronic diseases, however, this danger is very great. Von Noorden[1] has performed a number of exhaustive tests in different diseases of the stomach in order to determine the size of this deficit. He calculated the caloric value of the food eaten by the patient of his own free will; he found that in ulcer of the stomach, in chronic catarrh of the stomach, and in nervous dyspepsia an average of 21 calories pro kilo was introduced. This amount is too small, for we know that the human organism requires an average of about 34 calories when at rest and about 40 calories pro kilo and *pro die* when at work. The physician should endeavor, to the best of his ability, to cover the demand for calories in chronic diseases of the stomach. In some exceptional cases it will be found that this cannot be done without endangering the life of the patient.

There are also a number of diseases of the stomach in which the organ should be spared. This, of course, is self-understood, and need hardly be mentioned. It applies particularly to acute diseases of the organ. In the majority of chronic diseases, however, this is different, for here the chief indication for treatment is to maintain and raise the general nutrition of the patient and at the same time to remember that no food should be given that might directly damage the diseased organ. Honigmann[1] has formulated the therapeutic postulates in cases of this character as follows: "The patient should be systematically educated to take a diet that fulfils all the demands of his general metabolism and at the same time considers the peculiarities of the lesions existing." In this way the physiologic powers of the stomach that remain intact are fully utilized, the perversions of function are not increased, and, if anything, are utilized in such a manner that they aid the organism in its attempt to maintain nutrition.

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[1] *Loc. cit.*

The Quantity of Food to be Taken; Summary of the Most Important Articles of Diet.—The physician should determine not only the quality of the articles of food administered, but also their quantity.

The amount of food that a sufferer from some disease of the stomach should take will vary according to the general constitution of the subject, according to the character and the duration of the disease, and other concomitant circumstances. The exhaustive investigations of von Voit have given us a solid basis from which to calculate the demands of a healthy subject for food. According to von Voit, a healthy adult of average weight should ingest 100 gm. of albumin, 50 gm. of fat, and 450 gm. of carbohydrate in the twenty-four hours. Nowadays we are accustomed to express the nutritive value of different articles of food by their caloric value, and no longer express the quantities ingested in grams of albumin, fat, or carbohydrate; thus, for instance, we do not say that a healthy adult needs daily 100 gm. of albumin, 50 gm. of fat, and 450 gm. of carbohydrate, but we say he uses up 2720 calories. Of these—

$100 \times 4.1 = 410$ calories are contained in albumin.
$50 \times 9.3 = 465$ in fat.
$450 \times 4.1 = 1845$ in carbohydrate.

In order to calculate the caloric value of any kind of food, the number of grams of albumin that are contained in it are multiplied with 4.1, the grams of fat with 9.3, the grams of carbohydrate with 4.1; then the whole is added: 1 gm. of carbohydrate and 1 gm. of albumin furnish 4.1, and 1 gm. of fat 9.3, heat-units. From these figures we can readily calculate the caloric value of the food from the average number of grams of albumin, fat, and carbohydrate that it contained. The tables of König, Jürgensen, and others are a convenient aid in this calculation.

This method of expressing the caloric value of food is justified particularly, as we know, from Rubner's law of isodynamics, that the different articles of food can replace one another according to their caloric value; in other words, it is immaterial whether 410 calories are introduced into the organism as 100 gm. of albumin or 100 gm. of carbohydrate or 44 gm. of fat.

This law of Rubner is very important in the pathology of diseases of the stomach, for it is frequently necessary to limit the amount of carbohydrate in one form of secretory perversion, that of albumin in another form. The universal application of this law is, however, limited in certain respects. This is due to the fact that our organism is accustomed to a diet of a certain composition, so that it cannot tolerate any great deviation from this average for any great length of time. This applies particularly to albumin, and we know that the amount of albumin introduced cannot be allowed to fall below a certain value without seriously damaging the organism.

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[1] *Zeitschr. f. Krankenpflege*, 1894, No. 8.

A human being at rest demands about 35 calories pro kilo of his weight; a person performing light work demands about 40 calories. From this we can calculate that the caloric value of the food of an individual weighing 50 kilos is from 1750 to 2000 calories. In order, therefore, to select the correct amount of nourishment the weight of the patient must be known; it is well, therefore, to weigh every case of stomach trouble when treatment is begun, and again from time to time thereafter. In my clinic this procedure has been carried out for a great many years.

From a practical point of view it is very important to know that we are able to replace the albumin in the food by carbohydrates and possibly by fats. This can be done only, however, to a certain degree. It is a useful thing to know, because there may be a transitory intolerance to albumin, so that it may be desirable to reduce the amount of albumin in the food and to replace it by an amount of carbohydrate and fat that corresponds to the caloric value of the albumin omitted. In the case of stomach-diseases it is usually a difficult matter to give much fat because it is not well tolerated, consequently the deficit of albumin must usually be compensated by the administration of an excess of carbohydrates.

Von Noorden[1] has discovered another very important fact by exact experimentation. He found that the nutrition of any given case is not necessarily impaired if the peptic power of the stomach alone is reduced, and that the assimilation of the chief articles of diet is not necessarily reduced under these circumstances, provided that the food enters the intestine within a comparatively short time, and before fermentation and decomposition have occurred; in other words, the intestine can vicariously assume the function of the stomach as long as the motor power of the stomach remains intact. This is also seen in certain operations on the stomach by which the disease of the stomach proper is not affected, but by which the entrance of the stomach-contents into the intestine is facilitated. Cases of this kind show in a most conclusive manner that under certain conditions the intestine alone is capable of thoroughly digesting the food. Quite a number of cases are on record in which the peptic power of the stomach was completely lost, but in which a decided and rapid improvement in the general condition of the patient occurred as soon as an operation like the one outlined above was performed.

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[1] *Lehrbuch der Pathologie des Stoffwechsels*, 1893.

In cases of atony of the stomach in which the ingesta are not moved into the intestine as rapidly as is desired the intestine cannot vicariously assume the digestive rôle of the stomach. These observations were made chiefly in human subjects; they can readily be corroborated by animal experiments. Czerny,[1] de Filippi,[2] and Ogata[3] have performed such animal experiments and have shown that an animal is capable of assimilating the regular amount of food, even after the stomach has been completely excluded from the digestion of food, and that no increased putrefaction occurred in the intestine even after the disinfecting action of the stomach-contents was eliminated.

We have already stated that in the majority of diseases of the stomach the great loss of strength and the emaciation are due to an insufficient ingestion of food. This fact can be regarded as fully established nowadays. Our chief effort, therefore, in treating cases of stomach-disease should be directed toward raising the general nutrition without, at the same time, damaging the diseased organ; in other words, we should attempt to introduce the necessary amount of food into the body.

On the one hand, therefore, we are confronted by the problem how to select a diet that is qualitatively adapted to the individual case, and, at the same time, to spare the stomach as much as possible without, on the other hand, introducing so little nourishment that the general metabolism of the patient is perverted in the sense of deficient assimilation and normal disassimilation.

This goal can, of course, not be reached in the same manner in every case. We have already mentioned in our discussion of the quality of the food that the nature of the disease and the peculiarities of each individual case must be carefully considered. If the disease of the stomach is a serious one; if the introduction of food is difficult, we should never forget that a patient who lies quietly in bed requires about one-seventh fewer calories than a patient who is up and about. For this reason patients who are sufferers from severe disease of the stomach and in whom nutrition is very much impaired should give up their occupation for a long period of time and enjoy complete rest. Whether or not we regulate the diet in any case by introducing more albumin, more carbohydrate, or more fat to fit the nature of the disease, we should always attempt to introduce a sufficient number of calories in the diet. At the same time we should avoid overtaxing the diseased organ. In order to comply with the latter postulate we should administer food that cannot irritate the stomach either chemically or mechanically. Above all, the food should be thoroughly macerated and finely divided. In cases of this character the food, moreover, should be nourishing, concentrated, and very finely divided.

If the physician desires to obtain a two-fold result in cases of chronic disorders of the stomach, namely, to cure the disease and to raise the general nutrition, he must individualize strictly in each case, and he must consider the habits and the tastes of each patient that he undertakes to treat.

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[1] *Beiträge zur operativen Chirurgie*, Stuttgart, 1878.

[2] *Deutsch. med. Wochenschr.*, 1890, No. 40.

[3] *Arch. f. Anat. u. Physiol.*, 1883.

In cases that can take only a liquid diet—for instance, recent ulcers of the stomach—milk is the best food. It is true that a milk-diet alone will not cover the demand of the organism for food, even though it is administered in very large quantities (3 to 4 liters). The average nutritive value of good milk is about 590 calories a liter, so that 3 liters correspond to 1770 calories. The majority of patients, however, with stomach-disease will not be able to tolerate such large quantities of milk, particularly if milk is the exclusive article of diet.

Because of these considerations, modern physicians no longer advocate an absolute milk-diet for a long period of time. This was formerly one of the favorite prescriptions in a great variety of diseases of the stomach. Nowadays, this method of treatment is employed only in the first stages of ulcerative processes of the stomach; in all other cases the physician, as a rule, replaces a part of the ordinary diet by milk; in other words, he gives a combination of milk and other easily digestible food, particularly barley-water and the like. If the milk is given in smaller quantities, combined with some such article of food as barley-water, it is usually well tolerated, even in cases where an absolute milk-diet is not tolerated. There are other difficulties to counteract in attempting to feed certain patients with milk alone that are attributable in part to the peculiar coagulative properties of the milk, in part to individual dislikes of the patient. A mixture called fat-milk, which Gärtner recommends, seems very appropriate; this mixture consists of pure cow's milk from which a portion of the casein, which is hard to digest, has been removed by some mechanical process; a part of the lactose is thereby removed, but the loss is replaced by adding a small quantity of milk-sugar. According to my own experience, which, by the way, is not very extensive, this food is better tolerated by patients than cow's milk alone, and I attribute this chiefly to the decreased amount of casein. The milk may be better borne and more easily digested if a small quantity of soda, lime-water, or magnesia usta is added. In the majority of diseases of the stomach it is permissible to add some fine preparation of amylaceous food—for instance, decoctions of tapioca, maizena, Löfflund's flour, etc.

The nutritive value of buttermilk is very small, so that this article of diet is not appropriate in cases of stomach-disease. The same applies to whey. A preparation that is sold under the name of kefir is better than these two. Of the different forms of condensed milk, I think that Löfflund's condensed milk is the best. Coffee and tea contain no nutritive material, and need not be considered in this connection.

Other fluid articles of diet that can be administered are meat-broths or bouillon and beef-tea. Meat-broth can hardly be considered a suitable article of diet, as it has no nutritive value; beef-tea, however, has some nutritive value. At the same time, the latter preparation is more of a stimulant and an appetizer than an article of food. Meat-juice, if recently expressed, has some nutritive value, as it contains approximately from 6 to 7 per cent. of albumin. The same applies to the popular preparation, which is comparatively cheap, namely, Valentine's meat-juice.

Sufferers from stomach-trouble also relish and digest different kinds of soup containing chicken or calves' brains, that are finely macerated or forced through a sieve; also soups that contain a little meat solution.

Certain gelatinous articles of food consisting of gelatin, calves' feet, etc., are appropriate articles of diet in diseases of the stomach, for, on the one hand, they replace the albumin and fat, and, on the other hand, they are easily absorbed, so that they contribute very much to the general nutrition of the patient.

In cases in which a meat-diet is permissible the different kinds of meat should be selected from one of the scales that we have mentioned above. The digestibility of the meat will be very much enhanced if it is finely distributed, and much will depend on the form in which it is served and the manner in which it is prepared. In selecting the meat the age of the animal, the tenderness and the age of the meat, etc., must all be considered. The digestibility of the meat can be increased by a number of manipulations, as beating, grinding, scraping, chopping, etc.

The digestibility of eggs will depend altogether on the manner in which they are prepared; soft-boiled eggs are probably the best.

In selecting fish for cases of stomach-disease those kinds that contain little fat—as shell-fish, trout, pike, halibut, and carp—are most readily digested, whereas the fat varieties, as salmon, eel, herring, etc., are not so readily digested.

Among the carbohydrates we have a great variety to select from. In order that they be digestible, the digestive processes in the mouth and the intestine must be normal. It is necessary, therefore, that this class of food should be well chewed and carefully insalivated. If the carbohydrate food contains much cellulose, as is the case in cereals, they are not so readily digested. It is best, therefore, to grind them thoroughly before using them as food. If there is a tendency to stagnation in the stomach, so that there is danger of fermentation, the administration of carbohydrate food should be carefully supervised. Ordinary bread (rye bread or domestic bread which is prepared from fermented dough) is not a suitable article of diet for most cases of stomach-disease. The best form in which amylaceous food should be administered to such cases is Zwieback, toast, fine white bread, maizena flour, tapioca flour, oatmeal, and Löfflund's "Kindermehl." All these different kinds of flour can be prepared in different ways. Aleuronat flour, which Ebstein[1] has recommended for dyspeptics, is a suitable article of food, as it contains very much albumin (over 80 per cent.). It seems worth while to try this food in stomach-diseases.

A number of leguminous prepared foods contain much albumin.

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[1] *Deutsch. med. Wochenschr.*, 1892, No. 19.

They are manufactured in such a manner that they are finely divided. Among these preparations I can particularly recommend Hartenstein's leguminose, Liebig's maltoleguminose, and Knorr's different preparations. Roots and bulbs are usually devoid of much nutritive value. If potatoes are eaten, they should be given in fine distribution—for instance, as mashed potatoes. Sago and tapioca contain much starch and are useful articles of diet; they should be given in the form of soups or mushes.

All varieties of cabbage and of leaf vegetables are best omitted from the diet of stomach-cases, as they contain very much cellulose; the same applies to salads.

Fruit possesses so little nutritive value that it need hardly be considered. In the discussion of the different diseases of the stomach we will outline in what form fruit can be eaten in different diseases.

There is a great diversity of opinion in regard to the amount of fat that stomach-cases should be allowed to eat. As fat possesses a high caloric value and at the same time a small bulk, it should be of value in all cases in which it is desired to raise the general nutrition. An objection is frequently formulated against the employment of fat—namely, that it constitutes a chemical irritant. Von Noorden has investigated this question and has found that this is true only in a mitigated sense. I can corroborate his experience. It is true that many cases of stomach-disease have a decided aversion against fatty food, and it is also true that they frequently complain of gastric discomfort or of nausea after eating fat meat, fat gravies, etc. Very fat meat prevents the stomach-juice from thoroughly permeating the food. The so-called white meats, chicken and veal, contain little fat and are very soft, so that they are readily digestible and better borne than the red meats, with the possible exception of raw or scraped beef.

[According to American experience, veal is not so readily digested as beef. There appears to be a peculiarity in the behavior of individual stomachs in this respect; for while veal is very indigestible with many, it occasionally is well tolerated and with some appears to be easily digested.—Ed.]

The same may be said in regard to fat as in regard to other articles of diet—namely, that everything may depend on the manner of preparation. Even traces of some fat that has a disagreeable taste may spoil the appetite altogether, whereas, on the other hand, large quantities of fat that are administered in a suitable form are frequently very well borne. This possible disadvantage may readily be avoided if fat is given in the form of butter, together with some carbohydrate.

Every stomach-case will readily tolerate considerable quantities of butter if it is given with cakes, Zwieback, or fine white bread, and should experience no discomfort from its ingestion. In this manner from 50 to 100 gm. can readily be given in a day, and this amount of fat possesses a caloric value of from 465 to 930 calories.

Another good preparation is von Mering's chocolate. In this food the fat of the cocoa bean is emulsified by the addition of a certain percentage of free fat acid. This makes it more readily digestible and enables us to give the patient large amounts of fat in a more palatable form.

If it is desired to spare the diseased organ and at the same time to raise nutrition, the form of the fat and the quantity of the diet must be considered, and, in addition, the caloric value of the fat eaten. The only way in which to treat these cases successfully and in which the two chief postulates of treatment of stomach-diseases—namely, sparing the stomach and raising nutrition—can be reached is to calculate and consider carefully the caloric demands of each individual case.

THE CHEMICAL COMPOSITION OF THE MOST IMPORTANT ARTICLES OF DIET, ACCORDING TO KONIG AND OTHERS (GIVEN IN PERCENTAGES).

Meats and Fish.	Water.	Nitrogenous constituents.	Fat.	Nitrogen-free constituents.	Ash.
Beef, very fat	55.42	17.19	26.38	. . .	1.08
Beef, lean	76.71	20.78	1.50	. . .	1.18
Veal, fat	72.31	18.88	7.41	0.07	1.33
Veal, lean	78.84	19.84	0.82	. .	(0.50)
Mutton, very fat	47.91	14.80	36.39	0.05	0.85
Mutton, half fat	75.99	17.11	5.77	. . .	1.33
Pork, fat	47.40	14.54	37.34	. . .	0.72
Pork, lean	72.57	20.25	6.81	. . .	1.10
Hare	74.16	23.34	1.13	0.19	1.18
Venison	75.76	19.77	1.92	1.42	1.13
Chicken, fat	70.06	18.49	9.34	1.20	0.19
Chicken, lean	76.22	19.72	1.42	1.27	1.37
Young capon, lean	70.03	23.32	3.15	2.49	1.01
Duck (wild)	70.82	22.65	3.11	2.33	1.09
Partridge	71.96	25.26	1.43	. . .	1.39
Pigeon	75.10	22.14	1.00	0.76	1.00
Smoked beef	47.68	27.10	15.35	. . .	10.59
Smoked beef-tongue	35.74	24.31	31.61	. . .	8.51
Westphalia ham	27.98	23.97	36.48	1.50	10.07
Salted ham	62.58	22.32	8.68	. . .	6.42
Pomeranian goose-breast	41.35	21.45	31.49	1.15	4.56
Westphalia "Mettwurst"	20.76	27.31	39.88	5.10	6.95
Cervelat sausage	37.37	17.64	39.76	. . .	5.44
Frankfurter sausages	42.79	11.69	39.61	2.25	3.66
Liver sausage (first quality)	48.70	15.93	26.33	6.38	2.66
Smoked or boiled salmon	74.36	15.01	6.42	2.85	1.36
River eel	57.42	12.83	28.37	0.53	0.85
Fresh herring	80.71	10.11	7.11	. . .	2.07
Pike	79.59	18.34	0.51	0.63	0.93
Shellfish	80.97	17.09	9.34	. . .	1.64
Halibut	86.14	11.94	0.25	0.45	1.22
Carp	76.97	20.61	1.09	. . .	1.33
Oysters	89.69	4.95	0.37	2.62	2.37
Salted herring	46.23	18.90	16.89	1.57	16.41
Sardelles	51.77	22.30	2.21	0.45	23.27
Kippered herring	69.49	21.12	8.51	.	1.24
Caviar	41.82	31.36	15.61	2.23	8.98

It will be an easy matter for any physician to do this if he will utilize the well-known tables that we have and which give us the average

Milk, Butter, Cheese, Eggs.	Water.	Nitrogenous constituents.	Fat.	Nitrogen-free constituents.	Ash.
Cow's milk	87.42	3.41	3.65	4.81	0.71
Cream	65.51	3.61	26.75	3.52	0.61
Butter	14.49	0.71	83.27	. . .	0.95
Cream cheese	38.01	16.28	41.22	1.90	2.59
Milk cheese	43.87	34.99	11.37	5.40	4.37
Buttermilk	90.27	4.06	0.93	4.07	0.67
Whey	93.24	0.85	0.23	3.03	0.65
Hen's-eggs	73.67	12.55	12.11	0.55	1.12
White of hen's-eggs	85.75	12.67	0.25	. . .	0.59
Yolk of hen's-eggs	50.82	16.24	31.75	0.12	1.09

Vegetables, Bread.	Water.	Nitrogenous constituents.	Fat.	Sugar.	Nitrogen-free constituents.	Wood-fiber.	Ash.
Wheat bread	35.59	7.06	0.46	4.02	51.46	0.32	1.09
Rye bread	42.27	6.11	0.43	2.31	46.93	0.49	1.46
Fine wheat-zwieback . .	1.18	13.31	3.18	7.12	73.96	0.25	1.00
Cakes	9.60	11.00	4.60	. .	73.30	. .	1.50
Potatoes	75.48	1.95	0.15	. .	20.72	0.75	0.95
Asparagus	93.75	1.79	0.25	0.37	2.26	1.04	0.54
Cauliflower	90.89	2.48	0.34	1.21	3.34	0.91	0.83
Brussels sprouts	85.63	4.83	0.46	. .	6.22	1.57	1.29
Red cabbage	90.06	1.83	0.19	1.74	4.12	1.29	0.77
White cabbage	89.97	1.89	0.20	2.29	2.58	1.84	1.23
Spinach	88.47	2.49	0.58	0.10	4.34	0.93	2.09
Green peas	80.49	5.75	0.50	. .	10.86	1.60	0.80
Carrots	87.05	1.04	0.21	6.74	2.66	1.40	0.90
Noodles	13.07	9.02	0.30	. .	76.77	. .	0.84
Prepared oatmeal	8.89	11.49	6.32	. .	70.84	1.05	1.41
Leguminose	9.00	23.55	1.25	. .	64.05		2.15

Fruit.	Water.	Nitrogenous constituents.	Fat.	Free acids.	Sugar.	Other nitrogen-free extractives.	Wood-fiber.	Ash.
Apples	84.79	0.36	. .	0.82	7.22	4.81	1.51	0.49
Pears	83.03	0.36	. .	0.20	8.26	3.54	4.30	0.31
Plums	84.86	0.40	. .	1.50	3.56	4.68	4.34	0.66
Mirabelles . . .	79.42	0.38	. .	0.53	3.97	10.07	4.99	0.64
Peaches	80.03	0.65	. .	0.92	4.48	7.17	6.06	0.69
Apricots	81.22	0.49	. .	1.16	4.69	6.35	5.27	0.82
Grapes	78.17	0.59	.	0.79	14.36	1.96	3.60	0.53
Strawberries . .	87.66	0.54	0.45	0.93	6.28	1.01	2.32	0.81
Almonds	5.39	24.18	53.68	. .	. .	7.23	6.56	2.96
Hazelnuts . . .	3.77	15.62	66.47	. .	. .	9.03	3.28	1.83
Chestnuts . . .	51.48	5.48	1.37	. .	. .	38.34	1.61	1.72
Sugar, Honey.								
Cane-sugar . . .	2.16	0.35	. .	. .	93.33	3.40	. .	0.76
Beet-sugar, fine	. .	. . .	. .	. .	99.75	0.12	. .	0.13
Honey	19.61	1.20	. .	. .	73.72	5.28	. .	0.19

amounts of albumin, fat, and carbohydrate that the different articles of diet contain.[1] If these tables are consulted and if the different points of view that we have just enumerated are carefully considered, any practitioner should be able to prescribe a diet that is correct in quality and quantity and fully satisfies the caloric demands of the patient.

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[1] See, for instance, König's *Procentische Zusammensetzung der menschlichen Nahrungsmittel* (*Procentic Composition of the Food of Man*), 1888; Jürgensen's *Procentische Zusammensetzung der Nahrungsmittel* (*Procentic Composition of the Food*), 1888, etc.

The reader will find a variety of diet-lists in which the caloric value of the different articles of food is calculated in a number of works; for instance, in the excellent book by Wegele, *The Dietetic Treatment of Gastro-intestinal Diseases*, also in Biedert's and Langermann's *A Text-book of Dietetics: A Cook-book for Cases of Gastric and Intestinal Diseases*, etc. In discussing the different forms of stomach-diseases we will frequently have occasion to refer to such diet-lists. In order to enable my readers to formulate dietaries for themselves that are suitable to each case and that take the caloric value of the different articles of food prescribed into consideration, I subjoin a summary of the different constituents of the more important articles of food. In order to determine the caloric value of each kind of food the number of grams of albumin that it contains must be multiplied with 4.1, the number of grams of fat with 9.3, the number of grams of carbohydrate with 4.1, and then the three multiples obtained must be added. The sum will give the caloric value of the food prescribed.

In order to demonstrate the exact method of calculating the caloric value of different articles to the reader I add a few diet-lists that I copy from the report of Von Noorden,[1] quoted above. I have selected these particular ones because they seem especially well chosen.

I. CHIEFLY MILK-DIET, WITH THE ADDITION OF CARBOHYDRATES IN LIQUID FORM.

	Albumin.	Fat.	Carbohydrate.	Calories.
Very good milk 1700 c.c. . . .	70.2	66.3	69.7	1295
Soup from 30 gm. of tapioca flour and 10 gm. of albumose[2]	10.0	. .	30.0	164
Soup of 40 gm. of wheat flour with one part of milk, 10 gm. of cane-sugar, and 1 egg . . .	7.0	5.5	40.0	244
	87.2	71.8	139.7	1703

II. CHIEFLY MILK-DIET WITH THE ADDITION OF CARBOHYDRATES IN THE FORM OF MUSHES AND SOUPS.

	Albumin.	Fat.	Carbohydrate.	Calories.
Very good milk 1500 c.c. . . .	62	58.5	63	1056
Soup from 15 gm. of sago, 10 gm. of butter, 1 egg, 10 gm. of albumose	17	13.5	15	257
Mush made of 80 gm. of wheat flour, 1 egg, 10 gm. cane-sugar (two meals)	7	5.5	90	398
	86	77.5	168	1711

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[1] *Berlin. Klinik*, 1893, No. 55.

[2] 10 grams (2½ drams) of albumoses are contained in 90 c.c. (3 fluidounces) of Denayer's preparation, in 22 gm. (5½ drams) of Kemmerich's preparation, and in 30 gm. (1 ounce) of Koch's preparation.

III. MILK-DIET WITH SOLID FOOD MADE OF FLOUR, BUT LEAVING LITTLE RESIDUE, AND WITH MEAT-BROTH.

	Albumin.	Fat.	Carbo-hydrate.	Calories.
Milk 1250 c.c.	51	49	52	878
Meat-broth with 1 egg, 10 gm. of butter, 50 gm. of fine wheat bread (toasted or softened) . .	10	14	30	294
Cakes 70 gm., butter 15 gm. . .	5	12	50	337
Soup made from 30 gm. of tapioca flour, 1 egg, 10 gm. of butter .	7	14	30	282
	73	89	162	1791

IV. MILK WITH TENDER MEAT; SOLID FOODS MADE FROM FLOUR; BUTTER AND SOUPS.

	Albumin.	Fat.	Carbo-hydrate.	Calories.
Breast of young chicken [1] 100 gm. (weighed raw)	19.6	2.8	. .	106.4
100 gm. of mashed potatoes . .	2.0	4.0	20	127.4
2 eggs	14.1	11.0	. .	160.1
100 gm. of fine wheat bread (toasted)	7.0	0.5	55	258.8
30 gm. of butter	. .	23.0	. .	213.9
100 gm. of trout	19.3	2.1	. .	106.4
1250 c.c. of milk, and, in addition, soups	51.0	49.0	52	878.0
	113.0	92.4	127	1851.0

V. MORE ABUNDANT DIET—NON-IRRITATING.

	Albumin.	Fat.	Carbo-hydrate.	Calories.
Tender meat [2] 250 gm.	49	7.0	. .	266
Cocoa 20 gm.	4	6.0	8	105
3 eggs [3]	21	16.0	. .	235
100 gm. of Zwieback	8	1.0	75	. .
100 gm. of fine wheat bread . .	7	0.5	55	259
50 gm. of cakes	4	2.3	36	187
50 gm. of butter [4]	. .	44.0	. .	407
40 gm. of tapioca flour [5]	. .	. .	40	164
40 gm. of maizena flour	. .	. .	40	164
20 gm. of sugar [6]	. .	. .	20	82
1250 gm. of milk [7]	51	49.0	52	878
	144	125.8	326	2747

General Rules for Stomach-cases Before and After Eating.—Following our discussion of the quality and the quantity of the diet, it may be well to give briefly a few general rules in regard to the manner of eating; these should be observed by all healthy persons, and particularly by persons who are afflicted with some disease of the stomach.

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[1] Other lean meats, including fish and game, almost all have a similar composition, based on their weight in the raw state.

[2] Various kinds of meat, finely divided, raw, broiled, or roasted, with a little butter; hot or cold, given in two meals.

[3] One egg for cocoa, one for soup, and one raw or soft boiled.

[4] To be used with the starchy foods, the soup, and the cocoa.

[5] To thicken the soup made from stock.

[6] For the cocoa and corn-meal (maizena) pudding.

[7] Part to be taken alone; the remainder with the cocoa and corn-meal pudding.

The Act of Chewing and Digestion in the Mouth.—Every stomach-case should make it a rule to masticate thoroughly the food after insalivating it, and to grind the food as much as possible. All these rules seem self-evident, but everybody knows how often they are violated. It is necessary in the majority of cases to call the patient's attention to these matters repeatedly. A good set of teeth and painstaking care thereof are the fundamental conditions for good gastric digestion—in fact, of digestion in general. We need not here give the reasons for this. If a patient presents himself without teeth, the treatment of his case should be begun by ordering an artificial set. The experiments of Sticker[1] and Biernacki[2] have shown how important digestion in the mouth is for general digestion. These authors have shown that the function of mouth digestion is not only to grind up the food and to convert starch into sugar, but that the secretion of saliva exercises a direct and important influence on the secretion of gastric juice in the sense, namely, that the chemical and motor powers of the stomach are reduced if the food that is swallowed contains too little saliva.

The Sequence of the Different Meals.—A healthy person should arrange his meals so that they are taken at regular intervals. Even in a healthy person deviations from the ordinary mode of life, changing the time of eating, may cause disagreeable sensations. If this is true in healthy subjects, it is much more apparent in cases with diseases of the stomach. If the time of eating is changed, the length of time during which food is present in the stomach is also changed. An article of diet that would have been digested in a short time had it been eaten at the usual hour may require much more time for digestion if eaten at an unaccustomed hour. If the sequence of the different meals is irregular, therefore, the diseased organ is overtaxed. It should be one of the chief indications in the treatment of diseases of the stomach not to overtax the organ.

The nature of the disease and the character of the perversion of function will determine whether it is best to eat small meals at frequent intervals or larger meals at longer intervals. If a subject is a sufferer from atony, small meals should be given ; if a sufferer from hyperacidity, larger quantities of albuminous food seem to agree better. Here, too, certain exceptions are seen. The character of the disease alone does not always determine the frequency and the quantity of the meals ; the subjective sensations of the patient and other general conditions must be considered. In a number of diseases there is pain at the height of digestion ; in other cases, when the stomach is empty. Some patients feel very much better and the pain disappears as soon as they take some food ; in others again this occurs as soon as the stomach becomes empty. In the former case small meals should be given frequently ; in the latter, large meals at longer intervals.

One other point is of sufficient importance to warrant discussion here—namely, the temperature of the food and the drink.[1] The average person sins greatly in this respect by taking food and drink that is either much too hot or much too cold, or by eating some very hot article of food immediately after some ice-cold material, or vice versâ. Even in a healthy person such extremes of temperature may be detrimental to gastric digestion, and, of course, if the stomach is diseased, this is particularly the case. It is well known that iced water and ice-cold beer, if it is taken rapidly and when the body is overheated, may cause cardialgia and dyspepsia. Wegele[2] has called attention to the fact that in America there are a great many dyspeptics, and he perhaps correctly attributes this fact to the consumption of ice-cold drinks, iced water, etc., which are so popular in this country.

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[1] *Volkmann's Samml. klin. Vorträge*, No. 297.

[2] *Zeitschr. f. klin. Med.*, vol. xxi.

If the food is too hot, a great variety of disturbances may arise—in fact, the frequent occurrence of ulcer of the stomach in cooks has been attributed to the oft-repeated tasting of very hot articles of food; certain animal experiments have shown that very hot food may exercise a deleterious influence on the gastric mucosa. It is well known that drinks, particularly soups, coffee, tea, etc., are generally taken much too hot. Uffelmann,[3] and after him Wegele,[4] expresses the opinion that the best temperature should be one that is as near as possible to the blood-temperature—namely, 37° to 38° C. (98°–100° F.). At the same time they recognize that habit and the individual taste of the subject play a certain rôle in this regard.

Regulations to be Observed After Eating.—The question has frequently been agitated and discussed, whether stomach-cases should be advised to rest or to move about after eating. The answers to this question have been varied.

I believe that healthy subjects should be governed in this respect by their subjective sensations, but that in stomach-diseases the matter is different. We are never justified in applying rules and regulations that are correct in the case of a healthy subject to the case of a subject that is afflicted with a disease of the stomach. Experiments performed on animals can, of course, never be applied to human subjects without careful analysis.

Spirig[5] has performed a number of experiments in healthy subjects on the effect of rest and exercise on the acidity and the motor powers of the stomach. He performed his experiments on himself and on a second healthy person, and found that the acidity of the stomach is greater when the person is at rest than when he is moving about or performing some physical exercise, and that, inversely, the motor power of the stomach is better after physical exercise than after a period of bodily rest. Spirig, therefore, believes that in cases of hyperacidity the patient should be advised to move about after eating. Salvioli[1] arrives at the same conclusions. He, too, found that after fatiguing muscular exercise less hydrochloric acid was secreted, but that the propulsion of the food into the intestine was accelerated. Forster,[2] on the other hand, was unable to determine any difference in the time required for digesting certain articles of diet during rest or during exercise. Cohn,[3] in his animal experiments, found that the gastric digestion was slowed if his dogs were allowed to run about after eating.

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[1] On this point compare especially Uffelmann, "Die Temperatur unserer Speisen und Getränke," *Wien. Klinik*, 1887, No. 9.

[2] Wegele, *Die diätetische Behandlung der Magendarmerkrankungen*, 1893.

[3] Uffelmann and Munk, *Die Ernährung des gesunden und kranken Menschen*, second edition, p. 334.

[4] *Loc. cit.*

[5] *Dissertation*, Berne, 1892.

On the basis of all these results we can say that in a healthy, robust subject it is immaterial for gastric digestion whether he rests after meals or walks about. The whole question probably is a matter that must be decided by the subjective sensations of the individual. In weak and reduced subjects, on the other hand, particularly in sufferers from some disease of the stomach, this is altogether different. The only way to decide the question as to what is best for them is on the basis of clinical observation and experience. One of the most important regulations in diseases of the stomach is that the clothing should not be too tight after eating, so that the stomach is not compressed during the period of digestion.

The question whether the patient should simply rest or should try to go to sleep after eating has not been decided. Schüle[4] determined the acidity of the stomach while the subject he examined was asleep, and found that in a healthy subject who took a test-meal in the evening the acidity of the stomach was not greater during sleep than in normal conditions. He found, however, that the motility of the stomach was reduced during sleep. Schüle, therefore, recommends forbidding patients with hyperacidity to go to sleep after eating. I think this conclusion altogether too far-reaching; for, in the first place, the result seen in a healthy person cannot be absolutely applied to pathologic cases; in the second place, the results obtained from the examination of a test-meal taken in the middle of the day cannot be absolutely compared with the results that are obtained from the examination of a test-meal taken in the evening. For these reasons Schüle's experiments can hardly be applied to our question, chiefly because they refer to sleep at night, whereas we are attempting to determine the effect of sleep in the middle of the day on gastric functions.

Very violent physical exercise on a full stomach should, of course, be forbidden in all cases of stomach-disease. It seems that the best regulation to give cases of stomach-disease is to rest in a horizontal position after eating; this applies to all diseases of the stomach in which the tone of the gastric muscularis is reduced, to cases of ulcer of the stomach, and to all cases of irritation of the stomach; finally, to diseases of the stomach in which the general nutrition is at the same time very much reduced. The horizontal position removes a great deal of strain from the walls of the stomach, and prevents them from being too much distended and weighted.

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[1] *Arch. ital. de biol.*, 1892, vol. xvii., 248.

[2] *Handbuch der Hygiene*, 1882.

[3] *Deutsch. Arch. f. klin. Med.*, 1888, vol. xlvii

[4] *Berlin. klin. Wochenschr.*, 1895, No. 50.

If cases of atony of the stomach move about very much immediately after a full meal or after drinking large quantities of fluid, the stomach is pulled downward, and in this way the further development of atony is favored. Unfortunately, this regulation is indiscriminately given in a number of resorts in which diseases of the stomach are treated in a routine manner and where the attending physicians do not sufficiently specialize in individual cases. It may even occur that under these circumstances violent acute dyspeptic attacks are seen. I believe that Kussmaul's explanation of this occurrence is the correct one; he claims that under the circumstances outlined the duodenum may become mechanically occluded by the pyloric part of the stomach, which is distended and overweighted, occlusion occurring in such a manner that the heavy pylorus drags the duodenum downward and causes a narrowing of its lumen or a bending of the intestine at that point where the duodenum is attached to the spinal column (Fleiner[1]).

We need hardly explain the regulation that should be given to cases of ulcer—namely, that they should remain quiet as much as possible. In the majority of cases of this character the patients themselves feel that the distress they suffer is relieved by rest and aggravated by exercise. Another class of stomach-cases that should rest as much as possible are those in which the general nutrition is very much reduced. Rest in these cases reduces general katabolism and in this way acts beneficially.

A number of nervous diseases of the stomach call for an altogether different treatment. Here moderate exercise after eating usually agrees better than prolonged rest. Exercise must, of course, not be too violent nor too fatiguing.

The question whether *passive* exercise should be indulged in must, in some instances, be answered differently than the question in regard to active exercise. All patients with stomach-disease with the exception of certain nervous disorders should be forbidden to ride horseback. There is no objection to driving, although the benefits to be derived from such exercise are manifested more in the improvement of the general health than in any direct effect on the local disease of the stomach.

The chief regulation in regard to supper is that it should not be eaten too late and should not be too abundant; this applies particularly to cases in which the motor power of the stomach is reduced.

We need hardly mention that in all cases of stomach-disease the action of the bowels should be carefully supervised and regulated. It would lead us too far to discuss the different methods that are at the physician's disposal to accomplish this purpose. We refer for a careful description of all these measures to the sections on the different diseases of the stomach.

Methods of Feeding the Patient and at the Same Time Partially or Completely Sparing the Stomach.—In a number of cases, as we have seen, the administration of food is a difficult matter. This is due partly to the fact that the stomach is unable to digest certain articles of diet; partly that the food ingested is vomited, etc. Owing to these difficulties a number of methods have been devised to spare the stomach or to give it complete rest. Some authors advise administering certain articles of diet in a state that is directly assimilable and readily digestible; others attempt to administer food by other channels than the stomach. In the following paragraphs we will briefly discuss these methods.

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[1] *Samml. klin. Vorträge*, new series, No. 103, p. 76.

(a) Peptone, Albumose, and other Artificial Foods.—The albumin that is taken with the food is changed in the stomach or the intestine by the action of the gastric juice or of the pancreatic juice. The suggestion, therefore, seems a natural one to administer the primary digestion products—peptone and albumose—in the place of albumin itself. This seemed particularly indicated in all cases where the digestive power of the stomach or the intestine was reduced. The fact has been established beyond doubt that albumose and peptone may replace albumin, that the nitrogen they contain can be assimilated and built up into tissue-cells, and, in other words, that these substances are true tissue-builders. This knowledge seems to justify the administration of peptones and albumoses and different foods made from these substances in the place of albumin.

In reality, the word albumose should be used instead of peptone, because the majority of so-called meat-peptones really consist of albumose and contain only a very small quantity of true peptone, together with more or less soluble albumin and colloid material.

For practical purposes several other questions have to be considered. First of all, whether the administration of peptones and albumoses is really profitable to the patient and whether it possesses any advantages; secondly, whether we are allowed to give quantities large enough to replace albumin. In practice the indication is not to give small quantities of peptones or albumoses, together with large quantities of albumin, because we know that where large quantities of albumin can be borne there is no necessity for giving peptones or albumoses in addition.

The question, therefore, amounts to this, whether or not we are able to replace the albumin deficit by peptones in cases in which only very small quantities of albumin can be borne, and whether these peptones are capable of nourishing the patient, as well as chemically equivalent quantities of albumin.

All older experiments were more or less fictitious in this respect, and did not really answer this question, because the quantities of peptone given were comparatively small as compared to the quantities of albumin administered at the same time; here the albumin administered was in itself sufficient to maintain the nutrition of the patient, so that the exact rôle of the peptones in this respect could not be studied. Deiters[1] was the first investigator to determine whether albumose and peptone are capable of inhibiting excessive nitrogen excretion when they are administered with a quantity of albumin that alone is too small to do this. He found that the body is capable of maintaining its nitrogen equilibrium just as well with Denayer's albumose-peptone mixture as with meat, and that Denayer's albumose-peptone mixture is capable of replacing a meat-diet in certain cases of stomach-disease in which the condition of the patient does not tolerate a meat-diet.

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[1] Von Noorden, *Beiträge zur Lehre vom Stoffverlust des gesunden und kranken Menschen*, No. 1, 1892; and *Therapeut. Monatsh.*, 1892, p. 271.

Kuhn[1] performed similar experiments with so-called somatose in my laboratory, and found that somatose is capable of preventing a loss of nitrogen and of replacing albumin completely even if the rest of the food administered is altogether insufficient to maintain the nitrogen equilibrium of the body. If, on the other hand, a large quantity of nitrogen is given together with somatose, the assimilation of the latter substance is very poor; in fact, the food easily produced diarrhea.

On the grounds of all these experiments the question whether or not peptones and albumoses can replace albumin must be answered in the affirmative.

Another question is whether it is practical, or even necessary, to administer peptones and albumoses in large quantities. We need hardly discuss the popular prejudice existing among the laity that peptones are a particularly concentrated and strengthening food, and that only a few spoonsful may be taken in order to maintain nutrition. The fact that peptones are very expensive would, of course, be a detriment to their general employment; at the same time this obstacle would not be prohibitive, provided the advantages accruing from the employment of the peptones would compensate the financial sacrifice often demanded of the patient.

The crucial point in the whole argument is whether or not it is really necessary for digestion that albumin should be transformed into peptones or albumoses *in the stomach*. If this were really the case, the administration of peptones would naturally be indicated in all cases where there was insufficiency of gastric secretion. It has been shown, however, that albumin, even if it leaves the stomach unchanged, may be digested very well in the intestine, provided it leaves the stomach early enough and before decomposition and fermentation have occurred. This, of course, can happen only if no obstacle to the passage of the gastric contents into the intestine exists. It seems that the proteolytic power of the intestinal secretion, or, better, of the pancreatic juice, is greater than that of the stomach. The animal experiments of Czerny,[2] Ogata,[3] and others, as well as a number of clinical examinations, have demonstrated that albumin is assimilated and utilized to a great extent in the intestine, even though the function of the stomach is omitted. We see, therefore, that a loss of the peptic power of the stomach does not hinder the assimilation of albumin. The fact, therefore, that in any given case the chemical powers of the stomach are insufficient does not necessarily justify us in administering peptone preparations, nor should we expect very marked results from such treatment.

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[1] Kuhn and Völker, "Stoffwechselversuche mit Somatose, einem Albumosenpräparat," *Deutsch. med. Wochenschr.*, 1894, No. 41.

[2] *Beiträge zur operativen Chirurgie*, Stuttgart, 1878.

[3] *Arch. f. Anat. u. Physiol.*, Physiol. Abtheil., 1883, p. 89.

One great advantage, however, in the administration of these preparations is the avoidance of all mechanical irritation of the stomach. Cahn[1] has performed a number of experiments on dogs and has found that the belief that peptones are very readily absorbed in the stomach is not a correct one, and that they are neither absorbed more quickly nor transported into the intestine more rapidly than if they are formed from albumin in the stomach. Kuhn in his experiments also found that somatose was not better than meat or meat-powder in this respect. On the other hand, our experiments on human beings show that Cahn is not justified in applying the results of his animal experiments to human beings by stating that peptone constitutes an irritant to the stomach mucosa, causing an increased secretion of gastric juice. We could definitely determine that at least in the case of somatose this does not occur in human beings. Altogether, animal experiments do not seem to be very conclusive in this respect, because the presence of free hydrochloric acid cannot be demonstrated.

I think that another objection that is frequently made to the employment of peptone—namely, that this preparation causes diarrhea—is more valid. It seems that the proportion between the quantity of peptone and albumose given and the quantity of nitrogen in the other articles of diet eaten at the same time largely determines this factor.

It will be seen, from all that has been said, that it is hardly possible to formulate precise regulations for the administration of peptone and somatose. It has been proved that they are capable of replacing a meat-diet, so that in cases where the ingestion of meat, for one reason or the other, must be reduced, they have a sphere of usefulness. It must be remembered, however, that all peptones and albumoses do not possess the same nutritive value. Their nutritive value is determined not only from their composition, but from the facility with which they are absorbed, their taste, their durability, etc. Unfortunately, these preparations cannot be administered for a great length of time in sufficiently large doses, because the patients soon express an aversion for them.

The administration of peptones and albumoses seems to be especially appropriate in cases where the albumin-solving powers of the stomach are reduced or inhibited and where it is desired to avoid all irritation of the stomach. They are still more indicated in cases where digestion in the duodenum is disturbed. If the secretory powers of the stomach are reduced and its motor powers at the same time remain intact, digestion in the intestine can vicariously replace the deficiency of gastric digestion. In cases of this kind it appears that the administration of peptones and albumoses is not so necessary nor so urgent as in cases in which both the secretory and the motor powers of the stomach are greatly reduced.

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[1] Cahn, "Die Verwendung der Peptone als Nahrungsmittel," *Berlin. klin. Wochenschr.*, 1893, No. 24.

Cahn has formulated an objection against the administration of peptones in cases where the propulsion of gastric contents is impeded, particularly in cases of ectasy that are due to stenosis of the pylorus. This author claims that under these conditions peptone will remain in the stomach just as water would. I cannot consider this objection a valid one, for von Mering has shown that the stomach does not absorb any water and does absorb peptones. In cases of this kind the abundant administration of water is certainly contraindicated, but the administration of peptones is theoretically justified, so that the administration of peptones is a practical and a useful procedure.

It is true that prolonged administration of these preparations has been found to be impossible, for the reasons mentioned above. If it should be true—and this question remains to be investigated—that peptones cause an increased secretion of hydrochloric acid, their administration would be contraindicated in cases of hyperacidity and hypersecretion. In these forms of stomach-disease, however, there is really no reason why peptones and albumoses should be given, as the digestion of meat proceeds in the best imaginable manner. In cases of ulcer in which mechanical irritation should be avoided the administration of these substances might be tried, but even here they can very well be dispensed with. Whether or not the addition of peptone to the diet increases the secretion of hydrochloric acid must be determined by future clinical experiments. Whenever the administration of albumin is, for one reason or the other, insufficient, the administration of peptones and albumoses may be attempted in order to replace the nitrogen deficiency.

The most universally employed preparation of peptone and albumose is probably Kemmerich's peptone. Careful metabolic determinations have been made with Denayer's albumose-peptone (Deiters), with Ross's beer (Ewald and Gumlich[1]), with somatose (Kuhn[2]). Another peptone and meat preparation that deserves particular mention is the popular meat solution of Leube-Rosenthal. This food is prepared by chopping 1 kilo of beef into fine pieces, mixing it with one liter of water to which are added 20 gm. of pure hydrochloric acid, and boiling the mixture for ten to fifteen hours in a Papin pot; then the mass obtained is crushed, boiled for fifteen hours longer, neutralized with pure sodium carbonate, and evaporated to a mushy consistency. This preparation contains relatively small quantities of peptone, so that it cannot be compared in this respect with the other peptone preparations mentioned above. At the same time it is very easily digestible and is universally employed.

Among other artificial foods that belong to this group I may mention Weyl's peptone. One objection to this preparation is its disagreeable taste. Then there is Antweiler's peptone, Koch's peptone, Brand's meat preparations, Valentine's meat-juice, Debove's meat-powder, and others. The last-named preparation is made by roasting finely chopped lean beef on tin plates until it is completely desiccated; it is then powdered in a mortar.

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[1] *Berlin. klin. Wochenschr.*, 1890, No. 44.

[2] Somatose may be taken in milk, barley-broth, cocoa, or bouillon. Somatose is also sold in the form of biscuits (crackers) and of chocolate.

Huggard has described another method of preparing a meat-powder of this kind. Lean meat is cut into narrow strips, placed for a few minutes in hot fat or lard until its surface is browned, then placed on a sieve for a short time. The fat is allowed to drip off, and the meat is then dried for twenty-four hours in a baking oven at a moderate temperature. The meat after twenty-four hours is quite brittle, and can be ground to powder in a coffee-mill. Meat-powder of this kind can be utilized for a variety of purposes.

Liebig's soup may also be mentioned. A number of useful preparations of meat and milk are in the market; among them peptonized milk, Gärtner's fat-milk, and Voltmer's mother's milk.

(b) Rectal Alimentation and the Introduction of Fluids by Rectum.—In many diseases of the stomach it is necessary to spare the stomach altogether for a short or a long period of time. If the individual is robust and if the stomach is to be put at rest for a few days only, it is unnecessary to replace the ingestion of food by the mouth by the introduction of food by some other channel. If, on the other hand, the individual is weak and reduced, or in case it is necessary to stop the administration of food by mouth altogether for a long period of time, a substitute must be sought. There are a number of diseases in which the most carefully selected diet cannot be borne; there are others in which it is impossible for food to enter the stomach (stenosis of the pylorus or cardia); there are other cases again of stomach-disease in which it is an essential condition for the cure of the case that the stomach should be placed absolutely at rest for a long period of time. In order to enable the stomach to stop all work and in order to nourish the patient a variety of methods have been devised by which food can be introduced into the body without passing the pharynx or entering the stomach. The most natural suggestion, of course, is to use nutritive enemata in the place of food taken by mouth.

In the beginning these nutritive enemata were composed of meat-broth, milk, eggs, or any other article of food. The physicians who administered these did not take the trouble to determine whether or not these different articles of diet could be absorbed by the mucous membrane of the colon. Voit and Bauer[1] were the first to perform some exact experiments in order to elucidate this problem. They found, in their investigations, that in dogs the mucous lining of the rectum and the colon was unable to absorb a mixture of egg-albumen and pure water, but that it could absorb egg-albumen if it was shaken with a dilute solution of chlorid of sodium. Eichhorst[2] repeated these experiments and arrived at the same results.

Meissner[3] recommended the introduction of solutions of peptone. Voit and Bauer advised the injection of meat-juice obtained by expression of raw meat. Both of these ideas are theoretically better justified than the older methods. Unfortunately, the administration of the products recommended by Meissner and by Voit was prohibitive on account of the great expense involved. In addition, the enemata were difficult to prepare; and, finally, violent diarrhea was often seen after the administration of either.

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[1] *Zeitschr. f. Biol.*, 1869, vol. v.

[2] *Pflüger's Arch.*, vol. iv., 1871.

[3] *Zeitschr. f. rationelle Med.*, third series, vol. vii.

This was the status of the question of rectal alimentation when Leube,[1] in 1872, first advised the administration of meat pancreas enemata for rectal alimentation. Leube's idea was to transfer a part of the digestive process that normally occurs in the small intestine—namely, pancreatic digestion—into the large intestine, and to cause the formation of pancreas peptone in the rectum. He argued that the conditions for such a process were favorable in this part of the body, particularly as the temperature was constant and equal to that of other parts of the intestine. He introduced chopped pancreas mixed with albumin into the rectum.

Leube's prescription for preparing this meat pancreas enemata is as follows:

Take 150 to 300 gm. of scraped and finely chopped beef; to this add 50 to 100 gm. of pancreas (from a cow or a hog) that is free from all fat and also finely chopped. The two substances are placed in a dish, and about 150 c.c. of lukewarm water added; the mixture is stirred until it forms a thick, mushy mass. If it is desired to bring about the digestion of fat in the rectum, as well as the digestion of albumin, from 25 to 50 gm. of fat may be added to the above mixture. The best syringe to use for the injection is one devised by Leube himself; it is a pressure-syringe.

Leube first experimented on animals and found that this mixture is, in fact, digested in the large intestine, and that a large amount of nitrogenous material can be introduced into the body in this manner. Leube's meat pancreas enemata have also proved of value in clinical work. He himself reports one case in which the patient was nourished for six months exclusively by pancreas enemata.

It is astonishing that this method of feeding is not more universally employed. This is probably due to the rather complicated preparation of the food and the difficulties of obtaining the necessary material. Formerly I was in the habit of using meat pancreas enemata frequently, and over a long period of time, and I can, in every respect, corroborate the favorable results that Leube reports. In several of my cases I was enabled to keep patients alive for months with this treatment, and I remember one case with a stricture of the esophagus that was nourished for ten months by this means exclusively.

This complicated method of preparing rectal enemata has, moreover, been shown to be unnecessary by Ewald.[2] This investigator repeated Voit and Bauer's experiments in man with injections of eggs and found that white of egg is readily absorbed even without the addition of chlorid of sodium. He showed, further, that the albumin of eggs is absorbed as rapidly as the commercial peptones, and that they are capable of furnishing an amount of nutriment to the body that is equal to that furnished by a corresponding amount of peptones. It appears, therefore, that the mucous lining of the large intestine is capable of absorbing albumin that is not peptonized.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. x.

[2] *Zeitschr. f. klin. Med.*, vol. xii.

Huber[1] repeated all these experiments and found that emulsified eggs could be absorbed without any further addition, but he also found that the addition of a little salt before peptonization increased the absorption considerably. He even showed that twice as much could be absorbed under these circumstances. His method of peptonizing eggs was to add 200 c.c. to a 0.15 HCl solution to two eggs, to emulsify thoroughly the mixture, then to add 5 gm. pepsin, and to allow the whole to stand in the incubator at 40° C. for ten hours.

It may be considered established, therefore, that the addition of salt increases the absorption of albumin in the large intestine. The exact explanation of this phenomenon was not forthcoming until quite recently. Grützner is entitled to the credit of having first explained this phenomenon by experimental methods.

There is a popular belief to the effect that fluids injected into the large intestine are absorbed chiefly in that part of the bowel, and it is believed that the ileocecal valve prevents the entrance of fluid into the small intestine. Grützner,[2] however, has shown that particles of any material that are injected into the large intestine may, under certain conditions, even reach the stomach.

Grützner's experiments are, briefly, the following: Rabbits, guinea-pigs, and rats were starved for twenty-four hours; then an emulsion of animal charcoal or of finely chopped horsehair, or of sawdust, or of some other substance of this indifferent character, in physiologic (0.6 per cent.) salt solution, were injected into the rectum; four to six hours after the injection Grützner succeeded in finding some of these elements throughout the whole intestinal tract, even in the stomach, whereas the large intestine was found to be empty. If, on the other hand, an emulsion was made with distilled water or with hydrochloric acid solution, or with a solution of potassium chlorid in the place of sodium chlorid, the result was negative. He repeated his experiments in human beings, and found that if he injected an emulsion of starch in physiologic salt solution by the rectum, he could find the starch-granules in every microscopic preparation he made of the stomach-contents that was removed several hours later by aspiration.

Grützner is inclined to attribute this peculiar and surprising recurrent movement to the presence of sodium chlorid; he bases his assumption on the fact established by Nothnagel[3] that stimulation of the serous membrane of the intestine by sodium chlorid may produce antiperistaltic movements of the bowel. Grützner believes that this peculiar property of salt enemata to cause antiperistaltic intestinal movements may also explain the favorable effects seen after the administration of egg enemata that contain sodium chlorid. He expresses the belief that the whole mass injected is moved upward into the small intestine and is there absorbed in the normal manner.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xlvii.
[2] *Deutsch. med. Wochenschr.*, 1894, No. 48.
[3] *Beiträge zur Physiologie und Pathologie des Darms*, 1884.

If this assumption is true, the negative results of Voit and Bauer without salt, and the positive ones with salt, would be explained in a satisfactory manner. The same applies to the results of Huber that we have reported above. Different investigators, however, have formulated objections not only against the explanation offered by Grützner, but also against his experiments themselves. Christomanos[1] attributes the positive results reported by Grützner to something altogether different; he believes that the contents of the intestine is expelled by the animals, and that they lick it up. He prevented such an occurrence by appropriate measures and claims to have obtained altogether negative results.

These experiments again, however, are not altogether free from objection. We can hardly believe that Christomanos is correct when he claims that animals would lick up a fluid containing sodium chlorid, but not one containing hydrochloric acid, potassium chlorid, or simply water. Swiezynski[2] repeated Grützner's experiments in my clinic, and found that the forced conditions under which the animals of Christomanos had to live might have exercised a considerable influence on intestinal peristalsis. We cannot enter into the detail of Swiezynski's experiments; we are convinced that they were carried out with all necessary precautions, and their chief results may be summarized as follows: He determined positively that lycopodium introduced into the rectum of human beings or of dogs was capable of traveling upward as far as the intestine. Swiezynski could not, however, positively determine whether or not the addition of salt aided this upward movement. His experiments, however, make this assumption very probable. Swiezynski, therefore, arrived at the final conclusion that nutritive enemata do not benefit the system because they are absorbed in the large intestine, but because they reach higher portions of the intestine and are absorbed there. It seems, therefore, that Grützner's discovery throws a great deal of light on the fundamental experiments of Bauer and Voit.

Dauber[3] reports other negative results. He, too, like Christomanos, attributes the positive results exclusively to the habit of animals of licking the anus and of eating the ejecta. Dauber did not perform any experiments on human beings. Such experiments, it appears to me, would be particularly important in order to decide this question, for in human beings a positive result could not be interpreted in any other way than by assuming a recurrent peristalsis carrying the material injected into the rectum into the small intestine. I am not inclined to believe in antiperistalsis proper, but think it more probable that the surface epithelium of the intestine is chiefly concerned in causing the movement of material against the direction of normal peristalsis.

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[1] Christomanos, "Zur Frage der Antiperistaltik," *Wien. klin. Rundschau*, 1895, Nos. 12, 13.

[2] Swiezynski, "Nachprüfung der Grützner'schen Versuche über das Schicksal von Rectalinjectionen an Menschen und Thieren," *Deutsch. med. Wochenschr.*, 1895, No. 32.

[3] Dauber, "Ueber die Wirkung von Kochsalzklystieren auf dem Darm," *Deutsch. med. Wochenschr.*, 1895, No. 34.

Whichever explanation is furnished for the peculiar phenomenon we are discussing,—namely, the appearance of material injected into the rectum in the stomach,—the fact that this can occur is fully established by the experiments of Grützner and Swiezynski.

I might mention that some older reports are on record that speak in favor of Grützner's results. Trautvetter[1] succeeded in demonstrating the presence of certain fluids, which he injected into the rectum of living dogs, in the first portion of the small intestine. He used solution of potassium ferrocyanid.

Grützner and Huber's experiments certainly seem to indicate that the value of nutritive enemata is greatly enhanced by the addition of chlorid of sodium.

The special composition and method of preparation of different nutritive enemata may, of course, vary greatly. Nearly every writer on diseases of the stomach has formulated some prescription of his own. In the following I will give some of the more common preparations. Ewald recommends the following composition:

Two or 3 eggs are thoroughly mixed with a tablespoonful of cold water. A small quantity of flour is boiled in half a cup of a 20 per cent. solution of dextrose, and to this mixture a wineglassful of red wine is added. The egg solution is then slowly stirred into this mixture. Care should be taken that the solution is not so hot as to coagulate the albumin. The whole mass should amount to about a quarter of a liter.

In army and hospital practice 3 to 5 eggs are mixed with about 150 c.c. of a 15 to 20 per cent. solution of dextrose, and are either injected or allowed to flow into the rectum. It may be well, in some cases, to add a little starch solution or mucilage in order to make the mass more viscid, or a few drops of tincture of opium in order to mitigate any symptoms of irritation. I have personally found the following prescription of Boas very useful:

250 gm. of milk.
2 yolks of eggs.
A small quantity of salt.
A tablespoonful of red wine.
A tablespoonful of "Kraftmehl."

Jaccoud recommends as a rectal food:

250 gm. of bouillon.
120 gm. of wine.
The yolk of 2 eggs.
4 to 20 gm. of dried peptone.

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[1] Von Trautvetter, "Wie weit können Flüssigkeiten in den Darmcanal per anum hinaufgespritzt werden?" *Deutsch. Arch. f. klin. Med.*, vol. iv.

Rosenheim[1] recommends the following preparation: Either peptones (one or two teaspoonfuls) or well-stirred raw eggs; if carbohydrates are desired, 15 gm. of dextrose should be added. Fat is best administered, according to Zuntz, in the form of a fine emulsion of purified cod-liver oil made by shaking 30 to 40 gm. of cod-liver oil with a few teaspoonfuls of a 2.3 per cent. soda solution. All the solutions are poured together and filled up to 200 to 250 c.c. with lukewarm water.

Singer[2] recommends the following mixture: 125 gm. of milk, 125 gm. of wine, one or two yolks of eggs, a small quantity of salt, a teaspoonful of Witte's peptone. The mixture is prepared fresh, thoroughly stirred, and injected. Three such nutritive enemata can be given a day; I can hardly recommend giving more than three or, at most, four. This form of enema is, as a rule, well borne even if administered for a long time.

The foregoing formulas represent a few examples, and the composition of nutritive enemata can be varied in a great many directions. I am in the habit of employing 250 c.c. of milk, two or three eggs, with the addition of a small quantity of salt, and one or two teaspoonfuls of red wine. I cannot recommend the addition of peptones, as they easily irritate the intestine and may cause diarrhea. Wegele[3] also advises leaving out the dextrose, as this substance is too prone to cause decomposition. All nutritive enemata should contain a little salt. In conclusion I may mention that Revilliod and Zoppino[4] have quite recently recommended nutritive enemata with oil emulsion.

The following rules in regard to the method of employing enemata should be observed:

1. Before giving a nutritive enema the rectum should be thoroughly cleansed. The cleansing enema should be administered at least one hour before the nutritive enema. It is best to wait until the evacuation of the water has occurred.
2. The quantity of fluid injected at one time should generally not exceed a quarter of a liter.
3. After the administration of a nutritive enema the patient should be instructed to remain quiet for a time—at least for an hour.
4. If the large intestine is very irritable, it is advisable to add a few drops of tincture of opium to the enema or to introduce an opium suppository after the administration of the enema.
5. The enema should not be administered with a syringe, but with a funnel or an irrigator to which is attached a soft-rubber tube. The patient should lie either on his side or in an elevated dorsal position.

Nutritive enemata are indicated if, for some reason or other, it is desired to put the stomach completely at rest for a time; further, in cases where the stomach is no longer capable of holding sufficient food or no food at all; finally, in cases where the absorptive powers of the stomach are reduced or inhibited so that there is danger of inanition.

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[1] *Pathol. u. Therap. d. Krankheiten des Verdauungsapparates*, 1891.

[2] *Centralbl. f. d. ges. Therap.*, March, 1895.

[3] Wegele, *Die physikalische und medicamentöse Behandlung der Magen- und Darmerkrankungen*, Jena, 1895.

[4] *La sem. méd.*, 1894, No. 66.

The first indication is found particularly in recent cases of gastric hemorrhage; also in poisoning with certain cauterizing substances, during the first days after operations on the stomach, in certain very obstinate forms of ulceration of the stomach, in uncontrollable vomiting, in conditions where the stomach is exceedingly irritated, and the like.

It would probably be more rational *per se* to go still further and to place the stomach completely at rest in all cases of ulcer, in all cases where there is much irritation or where there are severe degrees of atony, and similar conditions. It would be very good treatment to administer all food by rectum in all these diseases. The utility of this plan seems so evident that it is surprising to find it so little employed. Only recently experiments have been made in this direction, and, as was to be expected, the results are eminently satisfactory. Donkin[1] in particular has treated a large number of cases of ulcer by rectal alimentation exclusively, in some instances for twenty-three days, and reports very goods results. Boas[2] also reports that, stimulated by these communications by Donkin, he treated 10 cases of severe ulcer with frequent recurrences and violent gastralgia by rectal alimentation exclusively, after having found that all other methods of treatment showed no results at all, or only very transitory improvement. He states that in the majority of these cases he produced a cure. In 4 cases rectal alimentation was carried on for ten days; in other cases, for fourteen days. Three to four injections were given every day. I have personally been in the habit of employing nutritive enemata in all cases of obstinate ulcer, particularly in the beginning of the treatment. In severe cases of atonic dilatation of the stomach I have also been enabled to obtain very good results by completely or partially putting the stomach at rest. It is surprising to see the improvement in these cases of atony and dilatation that may follow this plan even after a few weeks, and it is gratifying to notice how the general health of the patient improves and how diuresis increases. I can warmly recommend rectal alimentation, particularly in this form of stomach-disease. I do not, however, go so far as Rössler,[3] who recommends placing the stomach altogether or partially at rest in all cases of dilatation of whatever degree. I believe that the degree of atony and the nature of the disease must be carefully considered. All that is needed in many instances is to limit the amount of fluid introduced by mouth, and to increase the amount of fluid that is injected by rectum.

The second indication for the employment of rectal enemata corresponds more or less with the first one. It obtains if the stomach is unable to hold a sufficient amount of food, or, as in some cases, no food at all. This is seen in cases of stenosis of the esophagus and the cardia, in all cases of obstinate vomiting, in severe stenosis of the pylorus, etc. In all these conditions rectal alimentation is frequently the only method at our disposal for introducing food.

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[1] "On Gastric Ulcer and its Treatment by Enemata," *The Lancet*, 1890.

[2] *Diagnostik u. Therap. der Magenkrankheiten*, pt. i., third edition.

[3] *Wien. klin. Wochenschr.*, 1893, No. 40.

We have already mentioned that it may be very advantageous to the patient to introduce fluid by rectum in cases of simple gastrectasy. In all cases of ectasy the propulsion of the ingesta into the intestine is interfered with. The stomach itself does not absorb water; on the contrary, the entrance of certain substances into the stomach causes a secretion of water; this leads to a reduction of water in the tissues in cases of ectasy of severe degree, so that the patient suffers from thirst, even though an abundant quantity of water is introduced. In addition, the secretion of urine is decreased, and at the same time the ectasy of the stomach increases in severity as a result of the large accumulation of fluid in the stomach. In cases of this kind the administration of fluid through other channels than the mouth is thoroughly indicated, and of all the methods that we know of, the rectal one is the most convenient and the most appropriate one. By injecting water into the rectum we are enabled to prevent the loss of water in the tissues, and, at the same time, to spare the stomach; no large quantities of fluid accumulate in the organ, and the lost tone may be regained. It is evident that this method of treatment leads to better results in the pure atonic forms of ectasy than in those forms that are due to a stenosis of the pylorus, but even in the latter cases the administration of water aids, and is a rational, symptomatic method of treatment. Rectal injections of water alone may be given, or a little salt may be added to the enema. Von Mering,[1] Rössler,[2] and Wegele[3] particularly recommend the addition of salt. Wegele recommends giving half a liter of lukewarm water containing half a teaspoonful of salt two or three times a day. I am in the habit of giving smaller quantities—about a quarter of a liter—and gradually increasing the dose. Other additions besides salt have been recommended—thus, for instance, brandy (one to three teaspoonfuls). Fleiner[4] recommends a mixture of meat-broth and wine (two-thirds broth and one-third non-acid white wine) for its analeptic and hypnotic action. My personal experience with such injections leads me to recommend them warmly. At all events, the deficiency in the absorption of fluid that is seen in every case of ectasy of high degree and atony of the stomach, whatever their cause may be, should be compensated by the introduction of a sufficient quantity of fluid by rectum. In some cases—as, for instance, in purely atonic ectasy—the results observed will be astonishing, and even if the primary cause of the trouble—as, for instance, stenosis of the pylorus—is not remedied, the ectatic process, at least, may be arrested, and the disagreeable symptoms caused by the abstraction of water from the tissues be relieved. Among these symptoms I may mention emaciation, dryness of the skin, torturing thirst, decreased diuresis, obstinate constipation, and a number of other symptoms. Nutritive enemata, as well as injections of fluid, deserve much greater attention than they have obtained so far. They are an excellent method for compensating deficiencies in the digestive powers of the stomach, and a useful means of sparing the stomach wherever the organ needs rest.

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[1] *Verhandl. d. Cong. f. innere Med.*, 1893.

[2] *Wien. klin. Wochenschr.*, 1893, No. 40.

[3] *Münch. med. Wochenschr.*, 1894.

[4] "Erfahrungen über die Therapie der Magenkrankheiten," *Samml. klin. Vorträge*, new series, 1894, No. 103.

(c) **Subcutaneous Feeding.**—If it is impossible to administer food by the natural passages, or if the administration of food in this manner is found to be insufficient, rectal alimentation is usually employed. At the same time there are several other ways of administering food, and one of the best among these is the subcutaneous injection of nutriment. Menzel and Perco[1], in 1869, performed experiments in this direction on dogs. They introduced fat, albumin, and sugar subcutaneously, and demonstrated that liquid fats could be absorbed without causing any local or general reaction. They then extended their experiments to human subjects. A patient in Billroth's clinic, who was a sufferer from caries of the spinal column, received 9 gm. of fat subcutaneously. A tumor appeared at the point of injection about as large as a dollar, but disappeared after thirty hours. The same authors experimented with injections of cow's milk, simple syrup, and yolk of egg.

Karst[2] recommended the subcutaneous injection of defibrinated blood, and Camerer[3] injected blood-serum. These experiments have so far been extended to human subjects in only a few cases; thus, Landenberger[4] injected calves' blood, Krueg,[5] olive oil, Witthaker,[6] a mixture of milk and cod-liver oil. Witthaker's patient received 68 injections in all; on one day 8 injections were made and 124 gm. of material were given. Eichhorn[7] performed some very extended experiments in animals, with the injection of different substances, as peptone, diluted milk, cod-liver oil, etc. This investigator feels justified in drawing the conclusion from his experiments that the subcutaneous administration of the above-named articles of diet may replace the taking of food by the natural passages, and may maintain the nutrition of an animal without damaging it in any way.

However interesting these experiments may be, exact demonstrations are still lacking to show whether this method of administration can really replace the eating of food, and whether metabolic equilibrium can be maintained by subcutaneous feeding. Leube,[8] of recent years, has experimented in this direction and has attempted to answer this question. He has demonstrated beyond doubt that fat injected subcutaneously can be absorbed and assimilated, and can enter fully into normal metabolic processes. It would lead us too far to go into the details of his experiments. It is questionable whether the subcutaneous administration of food will ever become popular in cases where it is necessary to administer food by other passages than the natural ones, even though Leube's experiments seem to demonstrate positively that the fats, at least, can be fully utilized when administered in this way.

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[1] *Wien. med. Wochenschr.*, 1869, No. 31.

[2] *Berlin. klin. Wochenschr.*, 1873, No. 49.

[3] *Correspondenzbl. d. Württemberger Aerztevereines*, tome xliv., No. 3.

[4] *Württemberger med. Correspondenzbl.*, vol. xliv., No. 20.

[5] Abstract in *Wien. med. Wochenschr.*, 1875, No. 34.

[6] *Schmidt's Jahrbücher*, vol. clxxvii., No. 1.

[7] *Wien. med. Wochenschr.*, 1881, Nos. 31, 32, 33, 34.

[8] Leube, "Ueber subcutane Ernährung," *Verhandl. d. XIV. Cong. f. innere Med.*, 1895.

ON THE EMPLOYMENT OF ALCOHOL, TEA, COFFEE, AND TOBACCO IN DISEASES OF THE STOMACH.

Healthy subjects take alcohol, in the first place, as an article of luxury, then as a stimulant and as a strengthener. Within recent years a number of careful investigations have been published that explain the direct action of alcohol on the stomach. We will mention some of the most important of these experiments.

Gluzinski[1] has reported the most exhaustive investigations. According to this author, small quantities of alcohol slow the digestion of albuminates in the first stage of digestion; in the second stage, after the alcohol has disappeared from the stomach, the secretion of gastric juice is increased and prolonged, but the mechanical powers of the stomach are to a slight degree reduced. All this applies to a healthy subject. The impairment of the motor power of the stomach is so slight that it can hardly be considered detrimental. According to Gluzinski, small quantities of alcohol influence gastric disgestion favorably, inasmuch as they stimulate the secretion of hydrochloric acid. If large quantities are administered, this is not the case, for digestion is delayed, the mechanical functions of the stomach are hindered, and consequently the food remains in the stomach a longer time than normal. For all these reasons Gluzinski recommends the administration of small quantities of alcohol to be given a short time before eating, and claims that the exhibition of alcohol in this manner will favorably influence digestion.

The results seen from the administration of alcohol in pathologic cases were different. Here it was found that both in slight and in excessive acidity of the stomach-contents the administration of strong spirituous liquors did not aid digestion; on the contrary, digestion was considerably impeded in cases where there was a low degree of acidity of the stomach-contents, and, on the other hand, in cases of high degree of acidity, the irritation of the stomach mucosa was unnecessarily increased by alcohol. Gluzinski, therefore, recommends administering alcohol some time before eating in cases of this character, in which alcohol has to be given as an excitant.

Wolff[2] performed his experiments with brandy containing about 50 per cent. of alcohol and with beer. He showed that alcohol or brandy slightly stimulates the hydrochloric acid secretion of the stomach if administered in small quantities, but reduces the acidity of the stomach-contents and impedes the formation of peptones if larger quantities are given. Klemperer,[1] on the other hand, was unable to determine any increase of the gastric secretion after the administration of alcohol, but claimed to have seen a marked increase in the motor powers of the stomach.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxix. The experiments made some time ago in my clinic by Schellhaas, and reported in *Deutsch. Arch. f. klin. Med.*, vol. xxxvi., are not given in detail, as they consisted in the direct addition of varying quantities of alcohol to the gastric juice, and were not made on the patients themselves. It was shown by these experiments, however, that the addition of alcohol in considerable quantities tends to delay digestion.

[2] *Zeitschr. f. klin. Med.*, vol. xvi.

Wolffhardt[2] has performed some very exhaustive and very careful experiments with alcohol. According to this investigator, absolute alcohol in quantities of 15 to 30 gm. hinders the digestion of the amylacea and of meat; small quantities (30 to 40 gm.) of brandy containing 50 per cent. of alcohol, given either in single doses or in measured rations, caused digestion to be concluded in from thirty to fifty minutes sooner than it would have been concluded had alcohol not been administered. Larger quantities (60 gm.) of brandy of 50 per cent. seemed to impede the digestion of amylacea alone, but to increase the digestion of meat if taken during the meal; given in small quantities during the time of digestion, they retarded the conversion of meat by some thirty to forty minutes. Still larger quantities (90 gm.) taken during the time of digestion retarded digestion a great deal. According to Wolffhardt, red and white wines stimulate digestion both when they are taken during a meal and when they are taken before eating.

From all these experiments we learn that alcohol in small doses exercises no marked deleterious effects; on the other hand, no great advantage seems to accrue to digestion from its administration; for the slight effect it exercises on secretion, particularly if it is given in the form of wine, can hardly be considered important from a therapeutic point of view. If hyperacidity or hypersecretion exists, the use of alcohol is contraindicated. Larger quantities always act deleteriously and retard digestion.

In general, therefore, we may say that in diseases of the stomach we can get along very well without alcohol. In all conditions in which the stomach is irritated, as in ulcer, acute and chronic diseases of the stomach with increased secretion of gastric juice, alcohol is to be condemned. If in a case of this kind alcohol is indicated for other reasons,—for instance, if the patient is very weak,—it had better be administered in the form of an enema. It would probably be extreme to exclude alcohol from the diet of all cases of stomach-disease on general principles.

In many cases of stomach-disease a glass of wine taken before or during the meal stimulates the appetite, and in other forms again a glass of wine taken after meals seems to act very favorably. The former method of administering alcohol is particularly indicated in cases where gastric digestion is reduced, whereas the last-named form is more adapted to cases in which the secretion of gastric juice is normal or inclined to be excessive. It is self-evident that in deciding whether or not alcoholic beverages should be allowed the habits and the general condition of the patient should be carefully considered. The results seen in experiments on healthy subjects cannot be transferred without careful individualization to pathologic cases. Alcohol undoubtedly exercises a different effect on a stomach that is accustomed to the use of moderate quantities than on a stomach that has never been accustomed to alcohol. The concentration and the form in which alcohol is administered are also, of course, important.

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[1] *Zeitschr. f. klin. Med.*, vol. xvii., supplement.

[2] *Münch. med. Wochenschr.*, 1890, No. 35.

Beer is hardly to be recommended in diseases of the stomach. The relatively large quantity of fluid taken distends the stomach and dilutes the gastric juice, so that, for this reason alone, beer is not a proper article of diet for many stomach-cases. In addition, beer usually contains a considerable number of fungi, particularly yeast-cells, so that the drinking of beer may lead to the development of fermentative processes. At the same time beer agrees very well with certain classes of stomach-disease,—for instance, with simple hyperacidity,—and as this beverage contains a certain amount of nutritive material, it may be given in a limited number of cases; when allowed, it should be given in small doses. Ross's "Kraft-beer" contains still more nutriment, and seems indicated in cases that are reduced. Beer is contraindicated in all cases afflicted with atony of the stomach, with ectasy, with ulcer, and with hypersecretion.

All strong spirituous liquors, particularly drinks prepared with spices, are to be forbidden in stomach-diseases; only in those exceptional cases in which they seem urgently indicated for one reason or the other may they be permitted. Champagne, too, must be considered a beverage that is in general unsuited for diseases of the stomach.

It will be seen, from all that has been said, that in general alcohol should be stricken from the diet-list of a sufferer from any disease of the stomach; only in very rare cases will its administration be advantageous. It may occasionally act as a stomachic or as a stimulant to digestion. At the same time we cannot very well get along altogether without it. Alcohol is directly contraindicated in all diseases of the stomach where there is much irritation, also in all cases of dilatation of the stomach, particularly if the dilatation is a result of stenosis of the pylorus. It is true that the mucous lining of the stomach is capable of absorbing alcohol; at the same time von Mering has shown that this absorption of alcohol is accompanied by a greater or less secretion of water into the stomach, so that an accumulation of fluid occurs in the stomach that favors the further development of the dilatation. If in a case of this character the patient suddenly becomes very weak, or if, for some other reason, the administration of alcohol is indicated and called for, it had best be administered in the form of a wine enema. If given in this way the general indication of giving these cases as much fluid as possible by rectum is complied with.

[In America valuable experimental work has been done upon the question of the influence of alcohol upon digestion and nutrition by Atwater,[1] and by Chittenden, Mendel, and Jackson.[2] Atwater concluded that alcohol served as a food in the body, being oxidized and utilized as are ordinary food-substances. The latter mentioned experimenters found that alcohol increased the flow of the gastric juice both when taken into the stomach *per orem* and when introduced through a fistula into the intestine, the pylorus being ligated. They found the gastric juice thus secreted had a high proteolytic power. Alcohol was very quickly absorbed from the stomach. More fluid was obtained from the stomach a given time after a certain amount of dilute alcohol was introduced than was the case the same time after the same quantity of plain water. The motor activity of the stomach was delayed from fifteen to thirty minutes as compared with the water cases, when alcoholic fluid was given with the test-meal. Gastric digestion, on the whole, however, was not greatly influenced by alcoholic liquids. The experiments were made upon dogs.—ED.]

Coffee and Tea.—Coffee and tea possess no nutritive value, and when employed at all, they are, as a rule, given for their stimulating effect. The only nutritive value that coffee and tea possess is given them by the admixture of milk and sugar. Coffee and tea are, at the same time, not of equal value in diseases of the stomach. It is known that coffee does not agree as well with cases of stomach-disease as tea, particularly if there is some irritation of the organ. If, for one reason or the other, it is desired to give either coffee or tea, the latter should always have the preference. It will, however, never happen that the disease itself calls for the administration of coffee or tea.

Tobacco.—The physician is frequently called upon to answer the question whether or not a patient with stomach-disease should be allowed to smoke. It has never been definitely determined what effect smoking has on digestion. We know that it stimulates and increases the secretion of saliva; that it dulls the sensation of hunger; that it occasionally reduces the appetite, and that it accelerates intestinal peristalsis.

Wolff[3] has performed a number of experiments on the effect of nicotin on gastric secretion. His experiments, however, are not sufficiently numerous to allow us to decide this question definitely. It appears that nicotin is capable of stimulating the secretion of gastric juice, but that if given too frequently, it reduces gastric secretion.

In practice the general rule should be followed that patients with any stomach-disease should smoke as little as possible. In acute diseases of the stomach smoking should, of course, be altogether interdicted.

In chronic diseases of the stomach the matter is slightly different. In general it may be said that it will be by far better to stop smoking. There are, however, a great many patients who cannot very well get along without it, and with whom smoking in moderation undoubtedly agrees. More than a very few mild cigars should never be allowed each day, and they should be permitted only when it cannot be definitely determined whether or not the disease of the stomach and the appetite are unfavorably influenced by smoking. If smoking is permitted, the patient should be instructed to smoke after eating—that is, when the stomach is full—and never when the stomach is empty.

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[1] Bulletin No. 69, United States Department of Agriculture.

[2] *Amer. Jour. of Physiol.*, March 1, 1898.

[3] *Zeitschr. f. klin. Med.*, vol. xvi.

METHODS OF CURING AND RELIEVING DISEASES OF THE STOMACH.

1. DRUGS.

In the following section it will, of course, not do to describe all the drugs that have at different times been recommended for this or the other disease of the stomach. I will limit my discussion to the most important ones, and will briefly describe their action and the indications for their use. Among these remedies may be mentioned hydrochloric acid, alkalis, bitters, the so-called stomachics, and the artificial ferments. I will not enter into a discussion of sedatives, hypnotics, astringents, styptics, and other remedies that are ordinarily used in diseases of the stomach. I will refer to these in discussing the treatment of the different forms of stomach-disease. After describing the above-mentioned remedies I will briefly discuss the use of mineral waters in gastric troubles.

Hydrochloric Acid.—Formerly hydrochloric acid was prescribed in nearly all dyspeptic conditions. Of late, however, this remedy is not so universally employed, particularly since it has been shown that hydrochloric acid is not absent in every form of dyspepsia, as was formerly believed.

As late as 1883 Leube,[1] guided by direct experiments, expressed the belief that in nearly all cases of severe dyspepsia excepting in those that were of a purely nervous character there was a deficiency of hydrochloric acid. This statement by so competent an author seemed to ground the hydrochloric acid therapy, which had been in use for so many years, on a solid basis of facts.

In 1885, however, I[2] showed that this lack of hydrochloric acid is in many cases only apparent. I performed a number of comparative investigations both with the method recommended by Leube—*i. e.*, by stimulating the secretion of gastric juice by thermic irritation, when the stomach was empty—and by the method that is now in general use and has been employed in my clinic since 1879—namely, by aspirating stomach-contents for diagnostic purposes at the height of digestion and after the administration of a test-meal. It was found that if Leube's method was employed, the gastric juice obtained was never so active as the fluid obtained by our method. Because of these results, I felt justified in declaring that Leube's method was not so well adapted for determining the strength of gastric secretion as was my own. At the time when these experiments were performed we could also show that in a great many cases of dyspepsia the secretion of hydrochloric acid was increased. This was in direct contradiction to the generally accepted view.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii.

[2] *Ibid.*, vol. xxxv.

These results showed that the administration of hydrochloric acid was not justified in the majority of cases of dyspepsia; nevertheless, it was a long time before the medical world learned to restrict the universal administration of hydrochloric acid in these conditions.

The administration of hydrochloric acid can naturally be useful only in those cases where it can be demonstrated that a decrease in the production of hydrochloric acid has occurred. The only way by which this evidence can be furnished is to aspirate the stomach-contents after a test-meal; the stomach-contents can then be tested for free hydrochloric acid with Congo-paper or phloroglucin-vanillin. If these reactions are positive, the production of hydrochloric acid is sufficient, for a positive reaction demonstrates the presence of free hydrochloric acid in excess. In a case of this kind the exhibition of hydrochloric acid is not only superfluous, but may occasionally be detrimental.

If, on the other hand, the above-named color-reactions are negative, the administration of hydrochloric acid may be indicated; for a negative result shows that an insufficient amount of hydrochloric acid has been secreted; consequently free excessive hydrochloric acid cannot appear in the gastric contents; in other words, it is shown that the hydrochloric acid production is not normal. If this is the case, this deficit must be remedied. Hydrochloric acid was formerly, and is to-day, administered in the treatment of stomach-diseases chiefly to cover this defect. I do not believe, however, that the administration of hydrochloric acid always fulfils this indication.

Even if very large doses of hydrochloric acid are given in cases of subacidity, the deficit can hardly be completely compensated, for we can never give large enough quantities of the acid to cause the appearance of free acid in the stomach-contents in cases in which free hydrochloric acid was absent before. This is not surprising when we consider how much hydrochloric acid is needed for digestion. According to Honigmann and von Noorden,[1] 1 part of hydrochloric acid can saturate only 18 parts of albumin; 100 drops of dilute hydrochloric acid contain 0.8 gm. of hydrochloric acid, and this would be barely sufficient to digest 15 gm. of albumin.

In a number of books on diseases of the stomach different authors have mentioned a case reported by me[2] as a proof for the efficacy of hydrochloric acid administration. I reported that after administering relatively small quantities of hydrochloric acid (1.5 *pro die*) for two weeks to a case in which free hydrochloric acid had been constantly absent, it appeared in the gastric fluid in the morning; it was not found, however, in the evening even after the administration of hydrochloric acid had been continued for fourteen days. While these authors quote this case as evidence of the efficacy of hydrochloric acid, I personally am not inclined to consider it a proof of the value of hydrochloric acid administration, for we could not show that other factors—as, for instance, diet and methodic lavage of the stomach—were not concerned in this improvement. And at the same time we must not forget that after a test-meal, at the height of digestion, and later, free hydrochloric acid could never be demonstrated in this case.

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[1] *Zeitschr. f. klin. Med.*, vol. xiii.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xxxvi.

Reichmann and Mintz[1] report cases in which free hydrochloric acid was absent and in which it appeared after the prolonged administration of hydrochloric acid. They feel justified in concluding from this observation that the hydrochloric acid treatment may stimulate the stomach to an increased secretion of gastric juice or of hydrochloric acid. It will be a difficult matter, however, to decide in each individual case how much of the improvement must be attributed to the administration of hydrochloric acid and how much to the other factors of the treatment.

There is no doubt that in individual cases of subacidity the administration of hydrochloric acid may to a certain extent improve the digestion of albumin; at the same time the advantage gained by this will hardly be considerable, even if maximum doses of hydrochloric acid are administered. It is hardly probable that the internal administration of hydrochloric acid can stimulate the secretion of it by the stomach, as some authors have claimed; if in a few exceptional cases free hydrochloric acid was determinable in the stomach-contents after its administration, this does not demonstrate that hydrochloric acid produced this effect, for cases are not rare in which free hydrochloric acid is absent at first, and in which it appears after a few days of appropriate treatment. This is seen in cases in which no hydrochloric acid at all is administered. Du Mesnil's[2] experiments on healthy persons in whom the total acidity increased after the administration of hydrochloric acid may not be transferred to pathologic cases, and, on the other hand, they do not demonstrate at all that the administration of hydrochloric acid stimulates its secretion in healthy subjects. We can hardly agree with the above-named author when he claims that the total acidity of the stomach-contents was increased. The figures he published are as follows: In one of his cases the acidity of the stomach-contents before the administration of hydrochloric acid was 0.3; if 10 drops of hydrochloric acid were added to the test-breakfast, the percentage of hydrochloric acid in one case was 0.33, in another case 0.21 per cent.; if 15 drops were administered, it was 0.17; if 20 drops were administered, 0.21 per cent. Even in a healthy subject the values for the total acidity fluctuate within wide boundaries,[3] and only if a large number of experiments are performed can this source of error be eliminated. It is hardly credible, from what we have said above, that the addition of 10 to 15 drops of dilute hydrochloric acid to the food should cause an increase in the total acidity. At all events, we cannot see that any proof has been furnished to show that the internal administration of hydrochloric acid can stimulate the secretion of gastric juice. On the other hand, it is probable that hydrochloric acid administered internally in subacidity of stomach-contents is utilized in the same way as the hydrochloric acid secreted by the stomach mucosa. For this reason the administration of hydrochloric acid is indicated wherever *free* hydrochloric acid is absent in the stomach-contents. It must be remembered, however, that all cases in which free hydrochloric acid is absent are not pathologically identical.

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[1] *Wien. klin. Wochenschr.*, 1892.

[2] *Deutsch. med. Wochenschr.*, 1892.

[3] This is clearly shown by du Mesnil's own cases. Thus, in one case, the acidity was raised from 0.22 to 0.25 by the addition of 5 drops; the addition of 10 drops in one case raised the acidity to 0.34; in another, to 0.25; the addition of 15 drops, to 0.25; and the addition of 20 drops produced an acidity of only 0.21 per cent. These discrepancies cannot be regarded as due to an increase of acidity from the addition of hydrochloric acid; they are simply an expression of the normal variation.

In one case the deficit may be very slight, so that the amount of hydrochloric acid needed to cause the appearance of free hydrochloric acid is small; in another case, again, the deficit may be large. In the one case we are dealing with a mild disturbance; in the other, with some severe progressive process that leads to atrophy. If, in the former case, free hydrochloric acid appears after a short time, and if, in the second case, it does not appear, even though large doses of hydrochloric acid are administered, this need not surprise us. The absence of free hydrochloric acid is a symptom that can be interpreted in many different ways.

If it is desired to determine whether the administration of hydrochloric acid improves the digestion of albumin, a large dose should be administered—as large at least as the one I[1] recommended for the first time with particularly good results. Biedert[2] of recent years has employed similar doses in his own cases, and recommends their use. From 15 to 20 drops should be given about every half-hour or still more frequently, and the administration should be begun immediately after a meal. The exhibition of such large doses has this advantage, that large quantities of water must be introduced later on in order to dilute the hydrochloric acid; but even if such large doses as this are given, very little will, as a rule, be accomplished; that, at least, has been my experience.

I need hardly mention that hydrochloric acid should be taken through a glass tube in order to protect the teeth.

It appears to me that a much better method of treating these cases of subacidity would be to find ways and means by which the stomach-contents could be propelled more rapidly into the intestine, particularly as in almost all these cases a certain degree of atony is present. If the stomach-contents can be moved into the intestine, pancreatic digestion will vicariously assume the functions of the disordered stomach.

A second indication for the employment of hydrochloric acid was sought in its disinfecting and antifermentative properties. The stomach, without doubt, is intended to perform a disinfecting function, and it actually possesses the power of arresting a number of putrefactive processes and of abnormal fermentations. The hydrochloric acid of the gastric juice certainly performs an important rôle in this direction. On the other hand, it is exceedingly doubtful whether the few drops of hydrochloric acid that are usually administered to patients with gastric fermentation can hinder fermentation if it already exists. The ordinary dose of 6 to 8 drops of official hydrochloric acid contains about 1.12 gm. of hydrochloric acid. If this is introduced into the stomach after eating, it will be so diluted by the stomach-contents, even when the stomach contains only a few hundred cubic centimeters, that we can hardly expect any antifermentative or antizymotic action. In addition, we must remember that there are many forms of fermentation—as, for instance, yeast fermentation, which may continue very active despite the presence of an abundant quantity of hydrochloric acid.

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[1] Riegel, *Volkmann's Samml. klin. Vorträge*, No. 289, and *Deutsch. med. Wochenschr.*, 1886, No. 35.

[2] Biedert and Langermann, *Diätetik und Kochbuch für Magen- und Darmkranke*, Stuttgart, 1895.

However important, therefore, the presence of hydrochloric acid in the stomach-contents may be to prevent putrefactive processes and abnormal fermentation, it is impossible to check these processes by the administration of hydrochloric acid in cases where they are already in progress.

It is established to-day that one of the chief symptoms of stagnation of the stomach-contents is abnormal fermentation. In conditions of this kind methodic lavage of the stomach is indicated. Very frequently we will succeed in stopping fermentative processes by this method. In other cases where this simple treatment does not lead to the result, other antifermentative measures must be employed.

The administration of hydrochloric acid has also been recommended for a third purpose—namely, for its stomachic action. It is not clear how this action occurs; at the same time everybody who has had any experience will acknowledge that HCl does possess this power; in fact, this can be experimentally demonstrated. If it is intended to give hydrochloric acid as a stomachic, it should not be given after a meal, but about a quarter of an hour or an hour before eating. Large doses are not necessary, for smaller ones seem to answer the purpose as well. We frequently see the appetite improve, and the patients take more food if hydrochloric acid is administered in this way; we believe, therefore, that it is useful for this purpose, and we recommend it.

The administration of hydrochloric acid is contraindicated in all cases where hydrochloric acid secretion is increased. In cases where there is hyperesthesia for hydrochloric acid it is also contraindicated. Talma has described cases of this character. As hydrochloric acid determinations were not made, the possibility is not excluded that these were in reality cases of hyperacidity. This theory is more or less corroborated by Talma's[1] own statements, for he says that in a few of the cases in which the stomach was hyperesthetic for hydrochloric acid the stomach-contents disappeared more quickly than it would have disappeared from a perfectly healthy stomach. This speaks for hyperacidity, for we know that hyperacidity and increased motility of the stomach go hand in hand. [Hypoacidity is often, and anacidity generally, attended with increased motor activity of the stomach. Indeed, as has been shown, in cases of anacidity the stomach is prone to empty itself onward by means of the increased motor activity.

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[1] *Zeitschr. f. klin. Med.*, vol. viii.

There are also a few undoubted cases of gastric anacidity that present not irritation alone, but actual distress, after the administration of really good-sized doses of hydrochloric acid.—Ed.]

According to all that has been said, the administration of hydrochloric acid for therapeutic purposes has a much more limited field than is usually assumed. Judging from my own experience I am inclined to attach the greatest importance to the last-named action of hydrochloric acid—namely, its stomachic powers. Possibly it has some value as a gastric disinfectant. Finally, it may be given in order directly to aid digestion, although in the light of our present experience we can hardly expect marked results in this direction. [Pawlow has shown that the administration of hydrochloric acid increases the secretion of pancreatic juice, and this directly favors intestinal digestion. The fact may account for the improvement in a patient that seemingly follows the use of hydrochloric acid in cases of hypochlorhydria.

The knowledge of the fact that pepsin and hydrochloric acid secretions do not follow parallel courses seems to be of some particular value. It is true, as the author says, that pepsin is rarely absent from the gastric secretion, and yet there are cases in which the pepsin is sufficiently low to retard digestion. This may be true even when the HCl percentage is high, as, for instance, in some cases of ulcer. According to some of the later investigations, it would seem as though the pepsin and pepsinogen question had not been given sufficient attention. We have yet to explain the remarkable results obtained by Fremont, Lannois, and others in administering to patients the gastric juice of the dog, obtained through a fistulous opening, and a number of investigators have reported improvement in cases of hypochlorhydria by the use of this substance.—Ed.]

Alkalis.—The general administration of hydrochloric acid for therapeutic purposes was not introduced into medicine until Bidder and Schmidt[1] demonstrated that the acid of the gastric juice is hydrochloric acid. The administration of alkalis, on the other hand, has been in vogue since the days of antiquity, partly as a medicament, partly as mineral waters. In medical practice it has been known for a long time that alkalis exercise a very beneficial effect in certain diseases of the stomach, and are capable of removing certain symptoms. This was known long before experiments with alkalis taught us in what way they acted. Claude Bernard performed the first experiments on the effect of alkalis; he found that small doses of alkalis stimulate the gastric secretion in animals, whereas large doses produce a neutralization of the gastric juice. Leube[1] expressed the opinion a long time ago that the carbonate of soda found in Carlsbad water was capable not only of neutralizing the excessive acidity of the stomach, but also of stimulating the diseased mucous membrane, and causing a renewed and permanent secretion of gastric juice. Leube based these assumptions on certain experiments that he performed on dogs with gastric fistula. He introduced soda solutions into their stomach, and discovered the facts outlined above.

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[1] Bidder and Schmidt, *Die Verdauungssäfte und der Stoffwechsel*, Mita and Leipsic, 1852.

Jaworski[2] was the first to perform exact investigations in human subjects with the aid of modern methods of analysis. According to this investigator, small doses of alkalis are capable of neutralizing a certain portion of the acid in the stomach-contents. This effect, however, is soon counteracted by the reactive secretion of hydrochloric acid. This reactive secretion may be greater than the original secretion of acid. In small doses sodium bicarbonate and Carlsbad salts are a stimulant to the gastric function; in large doses they reduce the secretion of hydrochloric acid. If Carlsbad salts are used for a long time, even in small doses, the acidity of the stomach-contents and the secretion of pepsin are very much reduced, and if the salt is used too long, the secretion of hydrochloric acid and of pepsin may be altogether stopped.

Ewald and Sandberg[3] found that Jaworski's results did not coincide with their own experience in practice, and consequently undertook to repeat his experiments. They too found that Carlsbad water in small doses stimulates the stomach secretion, but they could not determine that the employment of this method of treatment for four weeks caused any decrease in the secretion of acid or of pepsin.

Du Mesnil[4] also performed a number of experiments, chiefly in persons with healthy stomachs. The experiments of this author correspond more or less with those mentioned above, and he too found that the administration of a certain limited quantity of bicarbonate of soda increased the amount of hydrochloric acid in the stomach-contents, and that large doses had to be given in order to cause a reduction in the total acidity. In contradistinction, however, to a *healthy* stomach, he found that in *hyperacidity* of the gastric juice the quantity of hydrochloric acid was immediately reduced. He found the same when he administered Carlsbad water instead of sodium bicarbonate.

All these results were obtained in normal individuals and do not necessarily apply to pathologic cases. This is shown, for instance, in the case of hyperacidity that Du Mesnil described, in which an immediate reduction of the quantity of hydrochloric acid followed the administration of bicarbonate of soda. Another objection to the application of the above results to practice is the fact that in these experiments alkalis were usually given together with the test-breakfast. Undoubtedly the results will be different if the alkalis are given with the meal, or on an empty stomach, or at the height of hydrochloric acid acidity. Finally, Du Mesnil employed the remedy only for a short time, so that his experiments give us no information whatever in regard to the action of alkalis when they are administered for a long time.

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[1] Von Ziemssen's *Handbuch d. spec. Pathol.*, vol. vii.

[2] *Wien. med. Wochenschr.*, 1886; *Deutsch. Arch. f. klin. Med.*, vol. xxxvii., and *Wien. med. Presse*, 1885.

[3] *Centralbl. f. d. med. Wissenschaften*, 1888.

[4] *Deutsch. med. Wochenschr.*, 1892.

Linossier and Lemoine[1] arrived at results that corresponded essentially with those of Du Mesnil. They could determine from experiments that bicarbonate of soda is an excitant for the gastric mucous lining not only in small, but even in comparatively large, doses, both when administered before and at the beginning of the meal.

On the basis of these results, they recommend the administration of this remedy some time before eating in all cases of insufficiency of the gastric juice. As the experiments of these authors, however, apply only to a ruminant animal, we are not justified in applying their results to pathologic cases in man without further investigation.

Spitzer[2] has performed a number of experiments that merit more attention and are more significant.

Spitzer performed his experiments in 18 subjects who were sufferers from disease of the stomach and who underwent the Carlsbád cure for four weeks. He determined the motor power of the stomach and the effect of Carlsbad salts. His experiments differ from those of all the other investigations performed before his time by the fact that his method corresponds to the actual conditions existing when a Carlsbad cure is administered, and by the fact that he does not give the remedy with the test-meal, like Du Mesnil. The subjects that Spitzer experimented on took the thermal water or the salt early in the morning, and the test-breakfast one hour later. The stomach-contents was not expressed for one or two hours.

In the course of these investigations it was found that in a cure of this kind the motor power was increased in the majority of cases and reduced in none of them. In the cases in which the stomach possessed peptic powers it was found that this power was not reduced after a four weeks' course. In mild cases of chronic catarrh a slight increase in the peptic power could be determined. The secretion of hydrochloric acid fluctuated within physiologic boundaries; if there was superacidity, the acidity of the stomach soon became normal, but was never reduced below normal.

In general, therefore, these experiments corroborate the findings of Ewald and Sandberg. Spitzer, however, found that, as stated by Jaworski, a great depression of gastric chemism is noticed, particularly in cases that are nervous and sensitive, but whose stomach is otherwise healthy. This condition is seen toward the end of the course, and particularly if the patients are, at the same time, living on a modified diet. Spitzer claims, however, that this condition is only transitory, and that as soon as the course of waters is stopped for a few days, normal gastric activity is recovered.

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[1] "Académie de Médecine de Paris," session of March 28, 1893; see abstract in *Münch. med. Wochenschr.*, 1893, No. 15.

[2] *Therapeut. Monatsh.*, 1894, No. 4.

Reichmann[1] has recently published a number of investigations that consider all the questions discussed above. It appears that his results vary in some of the most important points from those of the investigators that preceded him.

Reichmann employed the bicarbonate of soda alone in his experiments. Reichmann emphasizes correctly that the results obtained with the administration of mineral waters do not determine the effect obtained from the administration of pure alkalis, because the composition of these mineral waters is too complicated.

Reichmann, in the experiments that he performed on his patients, studied the effect of alkalis on an empty stomach and on a digesting stomach. He found that both weak and strong solutions of bicarbonate of soda introduced into an empty stomach caused an abundant secretion of gastric juice. He determined, however, that the amount of gastric juice excreted after the administration of bicarbonate of soda was not greater than if he administered an equal quantity of distilled water. He also saw that no particular influence was exercised on gastric secretion if the drug was administered at the height of digestion; so that the administration of bicarbonate of soda both in large and small doses a short time before a meal exercised no influence whatever. If, on the other hand, bicarbonate of soda was given immediately after a meal, both the total acidity of the stomach-contents and the quantity of free hydrochloric acid present in the stomach were markedly reduced, and this reduction he found was proportionate to the amount of bicarbonate of soda that he administered. Finally, he determined that the continued administration of bicarbonate of soda in very large doses (daily, 24 to 32 gm.) for a long period of time (four to eight weeks) exercised no influence on gastric secretion.

Let us recapitulate. Bicarbonate of soda exercises no influence on the secretion of gastric juice, whether it is given in small or in large doses, whether it is given in a single dose or is distributed in small doses over a long time. Reichmann, therefore, in contradistinction to the majority of other authors, arrives at the conclusion that bicarbonate of soda does not exercise any influence on the secretion of gastric juice, but that it does influence the gastric juice that is already secreted, in the sense, namely, of neutralizing and alkalinizing it, and thereby either neutralizing or alkalinizing the whole stomach-contents.

All that we have said above represents a brief summary of what we know to-day in regard to the action of alkalis. The results of different authors seem to be contradictory in respect to certain points. We must not forget that a number of the experiments reported are not applicable to sick persons, because they were performed on subjects whose stomach was healthy. Again, a number of the experiments reported by different authors, and particularly a number of the conclusions that these authors base on their experiments, can be made the subject of controversy. And, finally, a number of the experiments reported were performed under conditions that do not correspond to the conditions existing in actual practice.

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[1] *Therapeut. Monatsh.*, March, 1895.

No one, of course, will doubt that the administration of alkaline remedies is capable of neutralizing acid in the stomach. Reichmann's experiments have demonstrated this beyond doubt, and, in addition, our daily clinical experience teaches us the same. The question whether or not alkalis are capable of stimulating gastric secretion is not so simple to answer; some authors claim that it does, others claim that it does not. The majority of experiments that are on record are valueless for our purpose, because they were performed on healthy subjects. Even if we concede—and this proposition is by no means demonstrated—that alkalis may, under certain circumstances, stimulate the secretion of gastric juice in healthy subjects, this does not mean that the same must necessarily apply to pathologic cases. Du Mesnil's experiments show that the effect of alkalis is different in hyperacidity than in normal conditions. His analyses, moreover, did not give constant values, even when they were performed on healthy subjects.

If we leave aside the results obtained in healthy subjects, because we consider them altogether irrelevant to the question under discussion, we can still maintain that we are able to neutralize the acid that has been poured into the stomach by the administration of alkalis. In healthy subjects there is no reason why we should wish to neutralize this acid. In cases of hyperacidity, however, where too much acid is secreted, we do wish to accomplish this end. Any one can demonstrate in a very simple manner that it is, in fact, possible to neutralize the acid in a superacid stomach by the administration of alkalis; all he needs to do is to aspirate the stomach-contents before and after the administration of alkalis, and he will find that after the administration of alkalis the acidity is reduced. In fact, every sufferer from hyperacidity knows that some of his symptoms can be relieved by taking alkalis; as soon as a dose of alkali is taken, the disagreeable heartburn, acid belching, and pain stop.

In cases where an excess of hydrochloric acid is secreted alkalis should, of course, never be given on an empty stomach, but should be given after the excessive secretion of hydrochloric acid has occurred. It is worse than useless to administer alkalis at a time when no acid is present in the stomach.

In hyperacidity, therefore, alkalis should be given at the height of digestion—namely, when too much hydrochloric acid is present—or they may be given a short time before this period. They should never be administered before or during a meal, but always a short time after a meal. It is impossible to determine the exact time at which these remedies should be given—in fact, the best period will vary in individual cases. As a rule, the subjective symptoms and the complaints of the patient will furnish valuable clues. It is best to administer the alkalis a little before the time at which the distress of the patient is usually greatest.

In hypersecretion we must proceed a little differently. Here, too, it is best to give alkalis at the height of digestion, as hyperacidity usually obtains in this condition. In view, however, of the fact that in hypersecretion a large quantity of hydrochloric acid is present when the stomach is empty, and in view of the serious impediment that this condition constitutes for the digestion of amylaceous material, alkalis may also be given on an empty stomach.

The most appropriate form in which to give alkalis is as alkaline waters, particularly Carlsbad water or salts. The latter preparation contains a large percentage of Glauber's salts, and consequently promotes the subsequent evacuation of the material taken. It may occasionally be desirable to produce a more rapid evacuation of the stomach and bowels in some other way, and we need hardly mention that lavage of the stomach when the stomach is empty, followed by washing with alkaline waters, can be performed for the same purpose.

The administration of alkalis is frequently advantageous in cases of ulcer, not as a remedy for ulcer, but as a means of counteracting the condition of hyperacidity that is usually found in this lesion. In the acute forms of dyspepsia combined with increased hydrochloric acidity of the stomach-contents alkalis are also indicated. Solutions of alkalis and alkaline waters are good agents for washing out the stomach, because, on the one hand, they aid in dissolving and removing the mucus contained in the stomach, and, on the other hand, they aid in neutralizing the excess of acid products that may be present.

I have never been in the habit of employing alkalis in order to stimulate gastric secretion. As far as I can see, no proof has so far been furnished that it is possible to stimulate gastric secretion by the administration of alkalis, nor that they are capable of reviving gastric secretion after it had once stopped. The fact that the administration of alkalis seems to increase the acidity of the stomach-contents a little in healthy subjects proves nothing for pathologic cases, and, as a matter of fact, the increase observed in healthy subjects was frequently, though not always, so slight that it remained within the boundaries of physiologic fluctuations. In a few cases no increase whatever was observed.

At all events, a stimulation of gastric juice cannot be considered a characteristic property of alkalis. Any irritant, whether it be mechanical, chemical, or thermic, that is applied to the mucous lining of the stomach is capable of stimulating the secretion of gastric juice. Leube has shown that alkalis are not a chemical irritant of sufficient strength to cause a secretion of gastric juice in an empty stomach. I believe, therefore, that the therapeutic application of alkalis for the purpose of stimulating gastric secretion cannot be recommended on the basis of the experiments that have been reported so far.

I have never seen any untoward effects from the prolonged administration of alkaline remedies. There can be no doubt that some damage can be done if alkalis are administered when they are not indicated. No physician should prescribe alkalis unless he has determined by a preliminary examination that acid eructations, heartburn, and other symptoms are really due to hydrochloric hyperacidity and not to the presence of excessive organic acids. The administration of alkalis is indicated particularly in cases of pure hydrochloric hyperacidity. Of course, the exhibition of alkalis may give transitory relief even if the acidity of the stomach-contents is due to the presence of organic acids, but their administration is permissible only in acute dyspeptic conditions, and never in chronic dyspeptic states complicated by the formation of abundant quantities of organic acids.

I wish particularly to warn against the abuse of alkaline remedies without preliminary study of the precise indications for such treatment. At the same time I cannot agree with Rosenbach[1]; this author certainly occupies an extreme position when he says that the prolonged use of alkaline remedies is to be condemned under all circumstances. It is impossible to answer the question how long alkaline remedies may be given; at least, no statement can be made that is generally applicable. The matter must be carefully studied in each individual case. The stomach-contents should be aspirated from time to time and examined; the local symptoms and the subjective sensations of the patient will all have to be carefully investigated. We can merely say that as soon as a certain quantity of acid is present, alkalis should be administered. We have already shown that the values for acid obtained may mean hyperacidity in one case, and may mean nothing whatever in another.

The best alkaline salts to be administered are the following: Among the alkaline carbonates, sodium carbonate and sodium bicarbonate; among the alkaline earthy salts, magnesia usta and magnesia ammonia phosphorica, as recommended by Boas. It appears that magnesia usta is capable of neutralizing the largest quantity of hydrochloric acid, while ammoniomagnesium phosphate is nearly as active; about four times more sodium bicarbonate is needed to neutralize a given quantity of hydrochloric acid than of magnesia usta.

The majority of practitioners prefer sodium bicarbonate to sodium carbonate. This is due to the slightly irritating action of the latter preparation on the gastric mucous membrane and its disagreeable taste. The former preparation, on the other hand, has this disadvantage, that when given in larger doses it leads to the development of much carbonic acid gas and dilatation of the stomach; it is best, therefore, to prescribe a mixture of different alkaline salts.

I am in the habit of administering a mixture of bicarbonate of soda and magnesia usta in equal parts. In private practice a little of the ammoniomagnesium phosphate may be added. The fact that this salt is more expensive is not so important in private practice.

The dose varies in each case according to the degree of hyperacidity and the amount of food eaten at each meal. As a rule, half to one teaspoonful is sufficient. In some cases it is well to repeat the same dose or half this dose after a little time. A number of factors will determine whether or not other remedies should be added to this prescription. We will discuss the special treatment of the different diseases of the stomach further on.

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[1] *Münch. med. Wochenschr.*, 1894, No. 3.

I never employ alkalis in order to stimulate gastric secretion. The experiments reported above furnish no indication for this method of treatment. Those physicians who do use alkalis for this purpose recommend giving small doses about half an hour before each meal.

Carlsbad salts are frequently given in the place of alkalis and in the same diseases. They may either be used in the form of the natural sprudel salt or in the form of the artificial Carlsbad salt. A number of mixtures have been prepared by different authors similar to Carlsbad salts; one of these is the one described by Wolff,[1] consisting of 30 gm. of sulphate of soda, 5 gm. of sulphate of potassium, 30 gm. of chlorid of soda, 25 gm. of carbonate of soda, and 10 gm. of the biborate of soda.

Wolff recommends giving this mixture especially in cases of superacidity; he prescribes half a teaspoonful in half a glassful of lukewarm water three times a day on an empty stomach, two hours before dinner and two hours before supper. He claims to obtain very satisfactory results from this method of treatment.

In one case of typical hypersecretion combined with hyperacidity in which the acidity of the gastric contents at the height of digestion was 0.34 per cent. of hydrochloric acid, and in which more than 50 c.c. of gastric juice could be aspirated after fasting, the administration of this remedy for five days led to a reduction in the quantity of gastric juice that could be aspirated after fasting to 10 c.c., and a reduction of the hydrochloric acid at the height of digestion to 0.25 per cent.

I can testify to the fact that similar effects can be obtained by the administration of Carlsbad salts, provided that the diet is correct and other conditions are favorable.

It is a matter of great difficulty, of course, to determine the exact therapeutic significance of each measure employed in those cases where different therapeutic measures are used at the same time. In cases of hypersecretion no physician will limit himself to the administration of Wolff's salt or to Carlsbad salts alone, but he will, of course, prescribe an appropriate diet, will perform lavage, etc.

I might mention that biborate of soda has been included in Wolff's salt because Jaworski[2] demonstrated in some of his experiments that the prolonged use of borate of soda caused a reduction in the percentage of hydrochloric acid in the stomach.

I will not enter into a discussion of the different alkaline mineral waters at this point, as I devote a special section to this subject.

Bitters and Stomachics.—We know from practical experience that a number of remedies possess the property of stimulating the appetite; that others again can stimulate the secretory and motor functions of the stomach. The former property is usually attributed to bitter remedies; the latter, to so-called stomachic drugs. From a practical point of view these two groups can hardly be differentiated. Both remedies act by stimulating the appetite and promoting digestion. For the present, we do not know definitely just how this effect is brought about. In the case of some of these drugs it has been demonstrated that they stimulate the secretion of gastric juice; in others again that they stimulate the motor function of the stomach. In the case of a few we are possibly justified in assuming that they stimulate the glands of the mouth and in this way influence the secretion of the glands of the stomach indirectly. [This latter conclusion has been verified by a number of investigators.—Ed.]

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[1] *Zeitschr. f. klin. Med.*, vol. xvi.

[2] *Münch. med. Wochenschr.*, 1887.

In general, these remedies are administered when the appetite is low. Loss of appetite is seen chiefly in those diseases in which the secretion of gastric juice is reduced.

All the experiments that have been performed so far fail to reveal how these remedies act, and the results obtained are neither uniform nor satisfactory. Bokai[1] experimented on dogs that were starved for some time, and demonstrated that quassia and calumba were capable of increasing the secretion of gastric juice by a direct effect that they exercised on the mucous membrane of the stomach. Rensz[2] found that in artificial digestion tests *in vitro* the addition of bitters was capable of arresting artificial gastric digestion.

Huber[3] found that some of the bitter drugs in different strengths were capable of causing hyperemia of the intestinal tract and of stimulating intestinal peristalsis. Steckhoven,[4] on the other hand, failed to demonstrate that any of the bitter remedies that he tested could increase the secretion of gastric juice unless they remained in the stomach longer than an hour. Crelcow[5] found that the administration of extracts of bitter drugs to animals reduced the digestive powers of the gastric juice. Jaworski[6] experimented on human subjects and also arrived at negative results.

Reichmann[7] has performed a number of very careful investigations on the action of bitter remedies. He made his tests in patients with diseases of the gastro-intestinal tract, and not, as did his predecessors, with animals. This investigator selected two chief groups of bitter remedies for his experiments—on the one hand, pure bitters; and on the other, aromatic bitters. He selected four drugs from the first group—namely, herba centaurii, folia trifolii fibrini, radix gentianæ, and lignum quassiæ; from the second group he selected only wormwood.

Reichmann's experiments showed that the introduction of a bitter infusion into an empty stomach immediately caused much less stimulation of the secretory function of the organ than the introduction of an equal amount of distilled water. He was unable to determine any great differences between the different drugs that he tested, and found that the effect noted above occurred both in cases where the secretion was normal and in cases where it was reduced or increased. He found, however, that as soon as the bitter infusion left the stomach the gastric secretion was increased.

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[1] *Ungar. Arch. f. Med.*, vol. iii., Nos. 3, 4.

[2] *Ibid.*, vol. iii., Nos. 3, 4.

[3] *Ibid.*, vol. iii.

[4] *Weekbl. van het Nederl. Tydschr. voor Geneesk.*, 1887; abstract in *Schmidt's Jahrbücher*, vol. ccxix.

[5] *Or naczenii gorkich sredstw w piszczewarenii i uswajenii arotistych wieszczestw.* Petersburg, 1886.

[6] *Medycyna*, 1886.

[7] *Zeitschr. f. klin. Med.*, vol. xiv.

If the bitter infusion was administered together with the food, gastric digestion was more or less impeded, consequently the pieces of albumin that were removed from the stomach in these cases after a certain time were larger and thicker than in those cases in which albumin and water were given without the admixture of a bitter drug. It appeared, from his experiments, that the mechanical powers of the stomach were also slightly reduced if a bitter remedy was exhibited.

In those cases where the gastric secretion was about normal it could not be determined that the bitter remedy exercised any appreciable influence on gastric secretion while the stomach was digesting. In cases, however, of reduced gastric secretion he found that the administration of a bitter infusion increased this secretion. In cases, again, where no acid gastric juice was secreted at all, the bitter remedies were not capable of stimulating the mucous lining of the stomach to the secretion of gastric juice. In cases, finally, in which there was an excessive flow of gastric juice, the administration of bitter remedies caused an increase in gastric acidity. Reichmann's experiments also demonstrated that bitter infusions can be administered for a number of weeks without producing any changes in the function of a healthy or of a diseased stomach.

The results of these experiments led Reichmann to formulate the following principles in regard to the practical administration of bitter remedies. Bitter remedies should be administered only in those cases in which the secretory powers of the stomach are reduced, and in cases of this kind they should be administered about half an hour before eating.

Fawitzki[1] also recommends bitter tonics and agrees with Reichmann in advising their administration on an empty stomach and preferably a short time before eating. He claims that they act beneficially on the secretion of gastric juice in those cases where there is a reduced secretion of hydrochloric acid.

Wolff[2] performed a number of experiments with strychnin and condurango. Strychnin was given in the form of the nitrate of strychnin in doses of 5 mg. to 1.5 cg. (!) This investigator observed an increase of the hydrochloric acid production in 2 cases in which hydrochloric acid had been constantly absent. This stimulating effect of strychnin was observed to continue for several days after the administration of the remedy had been stopped.

Wagner[3] observed a similar favorable effect from the exhibition of tincture of nux vomica. According to Skjelderup and Duplay,[4] strychnin is said to exercise a favorable effect on the motor function of the stomach.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xlviii.

[2] *Zeitschr. f. klin. Med.*, vol. xvi.

[3] *Arch. gén. de méd.*, February, 1891.

[4] *Bull. gén. de méd.*, 1893.

In contradistinction to these results Wolff showed that the administration of cortex condurango in the form of the infusion or the maceration exercised no appreciable effect on the secretory or motor powers of the stomach either in a single dose or if the drug was administered for one or two weeks.

Finally, the experiments of Brandt[1] on dogs may be mentioned. This author found that the values for the gastric secretion observed after the administration of a definite quantity of peptone solution were not increased by the administration, at the same time, of certain spices, as white pepper, mustard oil, and peppermint oil, and that the values were constantly decreased to a considerable extent if spices are administered together with sugar solutions or watery solutions of iodid of sodium. On the other hand, he found that the administration of spices causes increased absorption in the stomach.

I have intentionally rendered all these experimental results in detail, so that the reader may formulate a judgment himself in regard to the value and the significance of the experimental evidence extant at the present time. However interesting the different facts elicited may be, they certainly fail to furnish a complete or a satisfactory explanation of the manner in which the different drugs act, especially as many of the results reported are altogether contradictory.

It would lead us too far to discuss all the different bitters and stomachics in this place. We will limit ourselves, therefore, to a discussion of two drugs that have been lauded within late years. I refer to condurango bark and orexin.

Condurango bark has been employed for many years among the aborigines of Ecuador as a popular remedy for carcinomatous and other chronic troubles. In Germany, Friedreich[2] first called attention to this drug in 1874. He reported a case of cancer of the stomach in which all the symptoms of the disease disappeared completely after the administration of condurango, so that the patient appeared to be completely restored to health. Since this report was published, condurango has been employed in thousands of cases of carcinoma, but has been unable to maintain its reputation as a specific for cancer of the stomach. In the same year in which Friedreich reported his observation I[3] expressed the opinion, based on a number of individual investigations, that condurango could be considered in no way a specific in cancer, but that the drug certainly possessed much virtue as a stomachic. This view is generally maintained to-day. A few observers, particularly Riess,[4] still praise the virtues of this remedy in carcinoma of the stomach and attribute particular powers to it. Riess examined 64 cases of cancer; in 17 of these a tumor could be felt and clearly outlined; he claimed that the administration of condurango caused a distinct and gradually progressive reduction in the size of these tumors, and that in 8 of the 17 cases the swelling disappeared completely or almost completely.

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[1] *Zeitschr. f. Biol.*, N. F., vol. xi., p. 277.

[2] *Berlin. klin. Wochenschr.*, 1874.

[3] *Ibid.*, 1874.

[4] *Ibid.*, 1887.

Three of these cases succumbed to some other disease, and in all some remnants of a previous tumor were found in the wall of the stomach, but no trace of malignant growth could be discovered, even microscopically.

These results, on first sight, are certainly startling; at the same time, they do not prove that the cases reported were really cured carcinomata. The same applies, so far as I can judge, to a case of carcinoma that was reported cured by Orszewczky and Erichsen.[1] I cannot agree with Riess that the action of condurango is more than that of a stomachic. It is a well-established clinical fact that carcinoma of the stomach may improve very much if the diet is well selected; this is seen particularly in cases in which the motor powers of the stomach are still intact. If we succeed in raising the appetite of our patient by the administration of condurango, his general nutrition will, of course, improve, and under these circumstances we need not be surprised to see the apparent size of the tumor decrease. The same applies to Friedreich's case, in which the autopsy revealed that the cancer still existed. The reduction in the size of the tumor was probably only apparent, and was simulated by the increase of the adipose tissue of the patient. This, of course, can happen only in cases in which the motor power of the stomach is still relatively good. Where ectasy exists, so that the stomach can get rid of the ingesta only with difficulty or cannot get rid of the food at all, things will be altogether different. Here the sensation of hunger and the appetite will be lost, despite the great reduction in the general strength of the patient; this need not surprise us, for whatever the origin of the sensation of hunger may be, one thing is positively established—that hunger is never experienced unless the stomach is empty.

We can possibly explain the fact that condurango in some instances improves the appetite, and in this manner improves the condition of the patient, while it does not improve the appetite in other cases, by assuming that the motor power is good in some cases, whereas it is deficient in others. From all this I feel justified in maintaining my original position—namely, that condurango is not a specific in cancer, but possesses the virtues only of an energetic stomachic. The best way to administer the drug is to give it in the form of a decoction of the maceration, as first advised by Friedreich—

℞. Cort. condurango,	15.0 parts;
Macerate for twelve hours with distilled water,	360.0 "
Then evaporate down until, when strained, it equals	180.0 "

D. S.—A tablespoonful twice daily.

I am in the habit of administering somewhat stronger doses of this decoction,—namely, 18 to 20 gm.,—and administer the drug three or four times daily about half an hour before a meal, in the dose of a tablespoonful. In case free hydrochloric acid is absent,—and that is the rule,—I add from 1 to 2 gm. of dilute hydrochloric acid. I avoid the addition of syrups. Instead of using the decoction of the maceration, the wine of condurango (teaspoonful doses), or the rather expensive fluid extract of condurango (in doses of from 15 to 25 drops), may be given.

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[1] *Petersburg. med. Wochenschr.*, 1876.

Another very popular stomachic that has been introduced of late years is orexin (phenyldihydrochinazolin hydrochlorate). This drug was first recommended by Penzoldt[1] in 1890. This investigator claimed that it produced the sensation of hunger and improved the appetite. The statements of Penzoldt were investigated by a number of clinicians, and I have studied the matter in my clinic.[2] Some authors report results similar to those of Penzoldt; others again state that they saw no effects, or only slight effects, from the administration of the drug. The majority of authors express the opinion that orexin is an irritant for the gastric mucosa.

In many cases of reduced hydrochloric acid secretion the administration of orexin seemed to cause an increase of this secretion. Penzoldt, in addition, found that the time of digestion was reduced.

This remedy is not universally employed so far. This is probably due to the fact that the results obtained are so uncertain; besides, the drug sometimes causes vomiting, has a burning taste, and is otherwise disagreeable to take.

Penzoldt,[3] therefore, has recently recommended basic orexin. He administers this preparation in capsules, and in this way does away with some of the disagreeable features. At the same time he advises taking the drug with a large quantity of liquid—about a quarter of a liter. Penzoldt, as a rule, gives 0.3 usually only once a day, preferably about 10 o'clock in the morning, with the second breakfast. He gives it for several days—usually for five days in succession. Among 31 patients who took the drug a distinct effect was observed in 23; Penzoldt, therefore, claims that basic orexin is a valuable stomachic; that it improves the appetite or, rather, is capable of restoring it; and that it is a useful drug in the milder cases of stomach-disease. The remedy is contraindicated in ulcer of the stomach, in hypersecretion, and in all conditions in which the stomach is irritated.

Holm[4] performed a large number of experiments with this drug and also claims that the basic preparation is preferable because it does not produce the disagreeable effects on the mucous membrane of the mouth and the esophagus that the hydrochlorate of orexin produces.

I will refrain from discussing other bitter remedies. I wish, however, briefly to mention creasote. This drug, as well as resorcin resublimate, is credited with the power of stimulating the appetite. According to the investigations of Klemperer,[5] however, the chief effect of this drug is manifested in an energetic stimulation of the motor function of the stomach.

In regard to strychnin, we have already mentioned that, according to

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[1] *Therapeut. Monatsh.*, 1890.

[2] Reichmann, *Deutsch. med. Wochenschr*, 1890, No. 31.

[3] *Therapeut. Monatsh.*, 1893.

[4] *Ibid.*, 1896, No. 1.

[5] *Centralbl. f. klin. Med.*, 1891.

Wolff, this drug possesses the power of stimulating the secretion of gastric juice and the motor function of the stomach.

We have also mentioned, in another place, that alcohol, particularly brandy, administered in small doses, may, under certain conditions, exercise a slightly stimulating effect on the secretion of gastric juice. If it is desired to bring about this effect by the administration of alcohol, it should be administered for a short time only, as the continued use of alcohol produces a craving for the stimulant, so that larger doses have to be given. As soon as this is done, the desired effect will no longer be produced.

The Digestive Ferments.—The digestive ferments are usually administered where they are deficient in the secretion of the stomach.

(a) **Pepsin.**—The administration of pepsin is only rarely indicated. For a long time the custom obtained to administer hydrochloric acid and pepsin in every case of obstinate dyspepsia. Nowadays, numerous experiments have taught us that wherever we find free hydrochloric acid there is also a sufficiency of pepsin in the stomach, and that even in those cases where hydrochloric acid is absent, pepsin is usually present. If pepsin itself is not present, pepsinogen at least will be found. Cases in which pepsinogen is also absent are exceedingly rare.

In general, therefore, the administration of pepsin in cases of stomach-disease is rarely indicated.

The preparation of pepsin that is made according to the directions of the German Pharmacopeia is not very active. According to this prescription, 0.1 pepsin is dissolved in 150 c.c. of water containing 2.5 c.c. of hydrochloric acid. This mixture must dissolve 10 gm. of bile albumin in four to six hours. It is self-evident that this does not answer all practical demands. There are, however, stronger preparations of pepsin—for instance, pepsinum concentratum of Jensen, Byk's pepsin, and other preparations. The so-called wines of pepsin cannot be recommended. According to Hugouenenq,[1] the addition of wine hinders the action of pepsin, and the administration of large doses of wine of pepsin may even hinder the albumin-digesting powers of normal human gastric juice (Werther[2]).

The best form in which to administer pepsin is the pure unmixed powder. The administration of this remedy is indicated only where a complete absence or a great deficiency in peptic ferment can be determined. If hydrochloric acid alone is absent but sufficient pepsinogen is present, all that is needed is to prescribe hydrochloric acid.

(b) **Papayotin and Papain.**—Preparations of papayotin and of papain are prepared from the milk-juice of carica papaya, a plant that is indigenous to the tropics. Würzt and Bouchut[3] were the first to test papain from this pure milk-juice of carica papaya for its chemical and physiologic properties. The latter author first administered it in dyspeptic diseases of childhood. Other authors since then have performed numerous experiments with the drug, among them Albrecht,[4] Rossbach,[1] Weeg,[2] Finkler,[3] Martin,[4] Chittenden,[5] and Hirschler.[6] All these authors agree that the ferment made from the milk-juice of carica papaya possesses digestive properties, and that albumin can be digested by extract of papaya in neutral, slightly acid, and alkaline solutions.

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[1] *Lyon méd.*, 1892.

[2] *Berlin. klin. Wochenschr.*, 1892, No. 27.

[3] *Compt. rend.*, 1879.

[4] *Correspondenzbl. f. Schweizer Aerzte*, 1880.

Papain and papayotin are very expensive, and consequently are not universally employed. Within recent years Sittmann[7] has again called attention to papain. This author directed all his investigations toward the therapeutic value of this drug. He used the preparation of papain that is manufactured by the firm of Böhringer and Reuss for his experiments, and claims to have observed most excellent results in different affections of the stomach in which the chemical functions of the organ were altered. He administered the drug as follows: 0.3 to 0.5 of papain were mixed with water to form a thin paste, and administered either in capsule or without a capsule after each meal. The meals consisted chiefly of meat. Sittmann claims to have seen very excellent results in a great variety of diseases of the stomach. In acute catarrh of the stomach he claims to have seen a favorable result after the administration of only two or three doses. In chronic catarrh of the stomach he claims that a cure was brought about within two weeks. In 3 cases of chronic dyspepsia in which an ulcer had existed at one time the appetite was recovered within a week. In 2 cases of cancer of the stomach the pain was reduced during the time that papain was administered; in one of these cases the digestion of meat was improved and a trace of hydrochloric acid that had been absent before was found. The most favorable results were obtained in dilatation of the stomach; here the administration of papain caused a more rapid solution of albumin and a more rapid evacuation of the ingesta.

The only clinicians who have so far repeated the investigations of Sittmann with the same preparation of papain are Osswald[8] in my clinic, and Hirsch[9] under Boas's direction. Osswald's experiments were directed merely to determining the physiologic action of the drug, and he found that papain is capable of converting albumin into albumose and peptone in neutral, alkaline, and acid solutions.

Papain has this advantage over pepsin that it is capable of exercising its effect in alkaline and neutral fluids so that it can continue its peptonizing function even after leaving the stomach. At the same time, the degree of peptonizing power and the rapidity of peptic action are less than those of pepsin itself. It appears, therefore, that papain is indicated only in those cases in which the secretory functions of the stomach are reduced.

Hirsch's physiologic experiments in the incubator show that papain can digest fibrin, raw, finely distributed meat, and solutions of egg-albumen most energetically, both in neutral and weak alkaline media, and that it can dissolve coagulated and finely divided egg-albumen in neutral and weak alkaline solutions, but that it cannot act on the last-named substances as rapidly as on the ones mentioned before.

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[1] *Zeitschr. f. klin. Med.*, vol. vi.

[2] *Dissertation*, Bonn, 1885.

[3] *The Therapeutic Gazette*, 1887.

[4] *Journal of Physiology*, 1884, 1885.

[5] See abstract in *Amer. Jour. Med. Sci.*, 1893.

[6] *Ungar. Arch. f. Med.*, vol. i.

[7] *Münch. med. Wochenschr.*, 1893, No. 29.

[8] *Ibid.*, 1894, No. 34.

[9] *Therapeut. Monatsh.*, 1894, No. 12.

Hirsch's therapeutic experiments are more important than his physiologic ones. He first used the drug in cases of stomach-disease in which peptic digestion was lost; here it was found that papain was not able to replace pepsin completely in those cases in which peptic digestion was more or less reduced, but that the peptonization of readily digestible albuminous food, particularly raw egg-albumen, milk, and raw meat, was greatly aided.

We have repeated the numerous experiments of Sittmann in our clinic, and were, unfortunately, unable to verify the striking results that this author claims from the administration of papain.

Papain preparations might be indicated, as far as we can determine, in those cases in which hydrochloric acid secretion is absent or very much reduced; thus, for instance, particularly in the later stages of chronic gastritis, in atrophy of the gastric mucous membrane, or in nervous anacidity. It might also be tried in acute cases of dyspepsia accompanied by anacidity or subacidity. In regard to the method of prescribing the drug and dose, it seems that the best way to give the remedy is in doses of 0.3 to 0.5 gm. several times after eating. Papain in tablet form has also been introduced.

(c) **Pancreatin.**—Fles[1] was the first to use the pancreas for therapeutic purposes in digestive disorders. His case was one of diabetes in which the stools contained very much fat and unchanged muscle-fibers. He immediately saw excellent results from the administration of pancreas. Engesser[2] was the first to prepare the useful preparation called pancreatin. This substance contains many impurities, but seems efficient and has been employed in many cases with good results. Since then a number of other preparations of powdered pancreas have been introduced, some of them, however, not very efficient; we are, to-day, in possession of a number of useful preparations of pancreas, among them the above-mentioned pancreatin of Engesser, and Merck's, Simon's, Witte's, and Schering's pancreatin.

Pancreas and the preparations made from pancreas are usually given in combination with carbonate of soda. Boas recommends a tablet consisting of pancreatin and sodium carbonate, of each, 0.5. From two to four of these tablets are to be given a quarter of an hour after eating.

Reichmann[3] calls attention to the fact that many of the commercial preparations of pancreas are frequently inactive, and advises the physician to prepare his own extract of pancreas as follows: Take a fresh pancreas from a cow, chop it fine, and place it in half a liter of alcohol of 12 to 15 per cent.; let stand for one to two days in a cool place and filter. The patient should be given a small wineglassful of this filtrate.

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[1] *Arch. f. d. holländischen Beiträge zur Natur- u. Heilkunde*, 1864, vol. iii.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xxiv.

[3] *Deutsch. med. Wochenschr.*, 1889, No. 7.

The administration of preparations of pancreas is particularly indicated in those cases in which the secretion of gastric juice is more or less reduced. If this ferment is given, pancreatic digestion occurs in the stomach ; this, of course, presupposes that the stomach-contents is neutral or only slightly acid, or that the acid of the gastric juice is partially or altogether neutralized by the alkali that is administered at the same time.

Pancreatin, therefore, acts in the same sense as papain, which we have described above ; it seems, however, that pancreatin is the more active of the two. For the present, the investigations in regard to the efficiency of pancreas are not sufficiently numerous to allow us to render final judgment in regard to the therapeutic value of this ferment in diseases of the stomach.

(d) The Salivary Ferment.—It appears that the saliva is rarely absent. Sticker[1] and Biernacki[2] have shown that the absence of saliva not only seriously interferes with amylolysis, but at the same time also with proteolysis. In cases in which the secretion of saliva is reduced jaborandi or pilocarpin may be administered, as these drugs are known to stimulate this function. Sticker did this in a case in which the secretion of saliva was almost arrested, and in which the secretion of gastric juice was at the same time very small. He reported good results from the administration of these drugs, for both the secretion of saliva and the secretion of gastric juice increased. At all events, cases of this character in which the power of the salivary secretion is quantitatively reduced, but the amount of fluid excreted is still more or less normal, are exceedingly rare. This has been demonstrated by Sticker in a series of investigations that he performed in my clinic and that have not yet been published.

There are a number of cases on record, however, in which the hydrochloric acid secretion of the stomach was so excessive and so continuous that it rendered the salivary ferment inactive even though in these cases the secretion of the salivary ferment was not reduced nor its powers insufficient. Boas recommends administering ptyalin or malt-diastase combined with alkalis in these cases. Personally, I have had no experience with ptyalin in cases of hyperacidity and hypersecretion. Saliva is not absent in these cases, but its action is interfered with by the excessive acidity of the stomach-contents. Cases of this kind should, at all events, be advised to chew their food carefully and for a long time. The administration of dextrinized amylacea may also be recommended. In order to diminish the acidity in these cases alkalis may be administered or the stomach may be washed out before starchy food is introduced.

In this way we frequently succeed in removing the causes that are responsible for the inactivity of the salivary ferment. Sticker has demonstrated that the administration of ptyalin or malt-diastase alone, without the administration of alkalis at the same time, is incapable of improving amylolysis. This was to be expected. At the same time it is demonstrated that alkalis alone exercise a particularly favorable influence. I do not see, therefore, why the administration of these ferments is indicated at all in cases of this character.

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[1] *Samml. klin. Vorträge*, No. 297.

[2] *Zeitschr. f. klin. Med.*, vol. xxi.

Mineral Waters.—Many patients with diseases of the stomach derive great benefit from a course of mineral waters. Cures of this kind are, of course, indicated only in chronic diseases of the stomach, and only certain forms of the latter are at all amenable to treatment by this method. There is a great diversity of opinion in regard to the choice of waters for different forms of stomach-disease; so far there is no scientific basis for the employment of any one water in any one disease.

The knowledge we possess is purely empirical. It has been found that in certain diseases of the stomach this, that, or the other form of mineral water seemed to benefit the patient. Only very few exact investigations into the effect of certain mineral waters on certain diseases of the stomach are on record.

Experiments performed in healthy subjects are not applicable, as a rule, to pathologic cases, chiefly because they are performed in a manner that does not correspond to the conditions existing when a patient takes a course of waters; consequently, too, the results obtained by this method of study furnish us no therapeutic clues.

As in all other diseases, the course of mineral waters should preferably be taken at the watering-place and not at the home of the patient. Undoubtedly, the regular mode of life, the change of surroundings, the good care that is exercised in all the details of life, the avoidance of all excitement, etc., are in great part responsible for the good effects that are frequently seen from a course of waters taken at a watering-place. Some clinicians deny this, and attribute all the good effects to the medicinal qualities of the water. The matter cannot, of course, be decided definitely, but we know that the employment of mineral waters is certainly more effective if taken at the watering-place than if the water or the salts it contains are administered at the home of the patient. If the surrounding conditions, the diet, the mode of life, and other similar factors are unfavorable, the course of waters, like any other mode of treatment, will not be successful. In stomach-cases, particularly, all these other factors are very important; the administration of waters or of salts at the home of the patient fails utterly if the patient does not, at the same time, lead a rational life, adhere to a suitable diet, and avoid all factors that might counteract the treatment. It is usually difficult to lead such a life at home; consequently the patient should be advised to seek one of the watering-places and undergo his course of treatment there.

The regulation of the diet is absolutely essential in every case of stomach-disease; if the patient is taking a course of waters, the regulation of the diet becomes still more important. The waters are one of the factors in the treatment, but not the only one; no good results from this treatment can be expected unless all the conditions are as favorable as they can be made. Whether we administer the remedies in the form of drugs or in the form of mineral waters, the mode of life of a sufferer from stomach-disease, and particularly the diet, should be most carefully regulated.

In many diseases the attending physician instructs the patient when to drink water, and how much to drink each time; he also gives him some general rules and regulations in regard to the diet during the course of treatment. In stomach-cases such vague instructions are not sufficient. A most careful dietary should be prepared, and the patient ordered to follow the instructions given him most rigorously during the whole course of treatment. The physician, in order to do this, must be thoroughly familiar with modern methods of examining and treating diseases of the stomach; unfortunately, this method of controlling the progress of a case is not universally employed in most watering-places; as a rule, certain articles of diet are forbidden, and that is all. The patient expects to be cured by the waters alone, and thinks that he can neglect the other factors enumerated; undoubtedly many failures can be explained on this basis. It is very much to be desired that patients who seek a watering-place should receive the benefit of better medical supervision in regard to their diet and the other factors that we have enumerated. The fact that in certain hotels articles of diet that are considered indigestible are not served is not sufficient; every detail of the diet should be carefully looked after by the physician, and the patient should be instructed to eat only those articles of diet that are prescribed. The regulation of the diet is as important a factor in the cure of cases undergoing a course of waters as the remedial effect of the waters themselves.

Four kinds of waters are employed in the treatment of diseases of the stomach—namely: alkaline saline waters, salt waters, alkaline waters, and alkaline muriatic waters.

The most important of these are the alkaline saline waters. They contain chiefly Glauber's salts, sodium chlorid, sodium carbonate, and some of them free carbonic acid gas. Some of these waters are warm, as Carlsbad, Bertrich; some of them cold, as Marienbad, Rohitsch, Sauerbrunn, Tarasp, Elster, Franzensbad. Carlsbad has the greatest reputation in stomach-diseases. That the waters of Carlsbad exercise a beneficial effect on certain diseases of the stomach was known long before direct investigations into the mode of action of these waters were performed. Jaworski,[1] and after him Ewald and Sandberg,[2] Spitzer,[3] and others have found that these waters exercise a direct effect on the secretion of gastric juice. The results obtained by these different authors do not correspond; Jaworski reports that the prolonged use of Carlsbad waters in the majority of cases causes a reduction in the acidity of the gastric juice and in the secretion of pepsin. Ewald and Sandberg were unable to find that the secretion of acid or of pepsin was reduced. Spitzer performed exact experiments in 18 patients with diseases of the stomach, and arrived at essentially the same conclusions

[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxvii.; *Wien. med. Presse*, 1888, Nos. 3, 4.

[2] *Centralbl. f. d. med. Wissensch.*, 1888, Nos. 16, 18.

[3] *Therapeut. Monatsh.*, April, 1894.

as Ewald and Sandberg; after a four weeks' course in Carlsbad the motor power of the stomach was increased in the majority of cases, never reduced; the secretion of hydrochloric acid fluctuated in either direction; if there was superacidity, normal values were soon obtained, never subnormal ones.

According to the experimental investigations that have been published so far, it seems certain that Carlsbad water is capable of reducing the overproduction of hydrochloric acid and that it is unable to interfere with the secretion of hydrochloric acid or of pepsin if given in ordinary doses. At the same time Jaworski's results should warn us from advising a Carlsbad cure for too long a time.

Carlsbad waters also seem to influence the secretion of bile and to possess the power of dissolving mucus and of stimulating the motor power of the stomach.

The results of all these investigations, combined with our practical experience, teach us to advise the administration of Carlsbad waters and other similar waters under the following conditions:

1. In round gastric ulcer, provided the stomach is not too atonic.
2. In so-called acid catarrh of the stomach, and also in all forms of dyspepsia with pronounced hyperacidity, provided the dyspepsia is not of a purely nervous character.
3. In dyspepsia in which the peptic power of the stomach is only relatively reduced; in cases of this kind the administration of Carlsbad waters should be carefully supervised.
4. In mild cases of atony.

The employment of Carlsbad waters is contraindicated in carcinoma, whether complicated by ectasy or not, in all severe cases of atony and ectasy, and in all cases of dyspepsia in which the production of hydrochloric acid is much reduced. A Carlsbad cure is not of great value in the majority of cases of nervous dyspepsia.

These statements represent the most important indications and contraindications; in the light of our present knowledge we are not justified in formulating any more precise distinctions.

The alkaline saline waters of Bertrich are similar to the thermal waters of Carlsbad; the former, however, are the weaker of the two.

Marienbad is the most important of the alkaline sulphate springs. This water contains about twice as much Glauber's salt as Carlsbad water, also more chlorid of sodium and more free carbon dioxid; it is more adapted, therefore, to cases in which there is intestinal atony. In general, Marienbad waters are less suitable for diseases of the stomach in which there is irritation than the warm waters of Carlsbad. If it is desired to administer Marienbad water in diseases of the stomach, it is better to give the water warm. The same applies to Rohitsch, Tarasp, Elster, and Franzensbad waters.

The waters of Rohitsch and Tarasp, like the waters of Marienbad, contain much free carbon dioxid; it is advisable, therefore, to warm these waters before giving them to stomach-cases. It is questionable whether carbon dioxid is capable of producing an anesthetic effect.

Even if this is true, as some authors claim, it is therapeutically unimportant.

The second group of mineral waters that is important in the treatment of diseases of the stomach are the pure salt springs. A number of clinicians have investigated the influence of salt on gastric digestion; the majority of these investigators have found that salt hinders gastric digestion more than it aids it. Alex. Schmidt,[1] for instance, found that the addition of 0.5 to 0.6 per cent. of salt impeded the solution of dialyzed albumin to a great extent. Marlé[2] administered small quantities of salt and failed to see any difference in the rapidity of digestion; if he administered large doses, however, he found that digestion was slightly retarded. Jaworski[3] found that the presence of salt neither stimulated the secretion of gastric juice nor caused a more rapid evacuation of the stomach-contents, but acted altogether negatively in both directions. Pfeiffer[4] determined that the addition of salt in a concentration of 0.24 to 0.4 per cent. hindered the digestion of albumin. Bikfalvi,[5] on the other hand, found that the administration of small doses of salt accelerated digestion, and that large doses retarded it. Leresche[6] carried out some experiments on a patient in whom a gastrostomy had been performed; he administered definite quantities of meat with and without salt, and found that whenever he gave quantities of salt that were not too small, the acidity of the stomach-contents was reduced.

Wolff's[7] experiments, as well as those of Reichmann,[8] showed that the acidity of the gastric juice was reduced by the administration of salt, and that this reduction was proportionate to the amount of salt given. Wolff draws the conclusion from these experiments that the favorable effect seen in diseases of the stomach from a course of salt waters, particularly of Wiesbaden and Kissingen water, is not due to the increased secretion of gastric juice, as was formerly believed.

On the other hand, Cahn[9] performed a number of experiments in animals by which he showed that hydrochloric acid disappeared from the stomach if the food contained little chlorin, and that the addition of salt caused hydrochloric acid to reappear in the stomach-contents. The practical experience of most clinicians does not correspond with the experimental observations of the investigators mentioned; in many of their experiments the conditions created did not correspond to the conditions that are seen in actual practice. Besides, the mineral waters used contained a number of other ingredients besides salt, so that some of the effects observed may be attributed to other substances than sodium chlorid.

Wiesbaden (Kochbrunnen, warm), Soden, Homburg, Kissingen, are well-known salt waters. To judge from practical experience, they stimulate the gastric secretion, the appetite, and possibly the motor power of the stomach. The waters of these different sources are indicated in cases of relatively recent gastritis in which the secretory powers of the stomach are reduced and in which there is an abundant secretion of mucus. They do not seem indicated in cases in which the secretion of gastric juice is increased, as in hyperacidity and hypersecretion, nor are they indicated in ectasies of high degree nor in carcinoma. Occasionally they do some good in carcinoma, provided atony and ectasy are not present. The good effects seen under these conditions are due to the improvement of the appetite; this improvement, while only temporary, aids the general nutrition of the patient.

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[1] *Pflüger's Arch. f. d. gesammte Physiol.*, vol. xiii.

[2] *Arch. f. exper. Pathol. u. Pharmak.*, vol. iii.

[3] *Zeitschr. f. Biol.*, 1883, 19.

[4] *Mittheilungen der amtlichen Lebensmitteluntersuchungsanstalt zu Wiesbaden*, 83, 84.

[5] Bikfalvi, *Ueber die Einwirkung von Alkohol, Bier, Wein, etc., auf die Verdauung*, Klausenburg, 1885.

[6] *Revue méd. de la Suisse romande*, 1884.

[7] *Zeitschr. f. klin. Med.*, vol. xvi.

[8] *Arch. f. exper. Pathol. u. Pharmak.*, vol. xxiv.

[9] *Zeitschr. f. physiol. Chemie*, vol. x.

The choice of waters in each individual case will not depend so much on the nature of the disease, as on the general constitution of the patient.

Wiesbaden Kochbrunnen is better than Kissingen, because it is hotter and contains less carbon dioxid. If Kissingen water is heated, it becomes more like Wiesbaden water—that is, it becomes milder, but at the same time never equals the latter.

The third group of mineral waters are the alkaline and alkaline-muriatic waters. The chief constituents of the former are sodium bicarbonate and carbonic acid; of the latter, in addition, sodium chlorid. The following waters belong to the former group: Bilin, Fachingen, Neuenahr, Giesshübl-Puchstein, Gleichenberg, Preblau, Salzbrunn, Vichy; and the following ones to the latter: Ems, Selters, Tönisstein, and others.

Simple alkaline waters are indicated in cases in which there is an overproduction of hydrochloric acid; this is due to the fact that they contain much sodium bicarbonate.

In practice they are frequently employed in this form of stomach-disease and have been found useful. The warm waters, as Vichy and Neuenahr (the former contains four times as much bicarbonate of soda as the latter), are particularly good. We know from experience that in hyperacidity and hypersecretion warm waters agree better with the patient than the more irritating cold ones; it is customary, therefore, to warm the cold waters before administering them to cases of this character.

It is not yet determined what rôle free carbon dioxid plays; only a few experiments have so far been performed on the effects of carbon dioxid on the gastric and intestinal functions, and the results obtained are neither uniform nor decisive.

The alkaline muriatic waters contain sodium chlorid in addition to carbon dioxid and bicarbonate of soda. In most of the waters the percentage of salt is relatively small. It appears that they are indicated in chronic catarrhs of the stomach, in mild degrees of atony, and in secondary catarrhs.

Bitter waters are hardly ever called for in the treatment of primary genuine affections of the stomach. The same applies to the simple aërated acid waters whose effect is due to the carbon dioxid they contain.

These waters are taken by many patients on account of their agreeable taste and their refreshing effect; some clinicians go so far as to claim that they stimulate the appetite. It has not been shown so far that they possess any other power.

It will be seen, from all that has been said, that no precise indications exist for the employment of different waters; so far we have been forced to rely on empirical observation alone, and the results obtained are very unsatisfactory. Occasionally it will be seen that the same form of stomach-disease improves if different waters are administered. We must never forget that in these cures many other factors, like the regulation of the mode of life, the avoidance of all excitement, the regulation of the diet, life in the open air, etc., contribute as much to the good results obtained as the chemical constitution of the waters themselves. For these reasons the selection of a watering-place should not be made from the nature of the disease of the stomach alone, but from the general constitution of the patient. I refer to Braun's[1] splendid work on balneotherapy for the more careful elaboration of this subject.

Climatic cures are not indicated in diseases of the stomach proper. In certain nervous affections of the organ they may do some good; at the same time sea-bathing or a sojourn in the mountains or other climatic cures may be indicated in diseases of the stomach, not so much in order to treat the disease of the organ itself, but in order to raise the general health of the patient; climatic cures and sea-bathing are particularly useful in nervous diseases of the stomach. In selecting a climate the general condition of the patient should be considered more than the local trouble.

Within recent years a number of sanitaria have been opened for the treatment of this group of diseases; this is certainly a step in advance.

Sanitarium treatment is, of course, indicated only in those forms of disease in which certain methods of treatment are to be employed that are difficult of execution at the home of the patient; in diseases of the stomach this is more frequently the case than in any other group of diseases, for in stomach-diseases the diet should be most carefully selected and the patient should be under the continuous supervision of a physician who is thoroughly familiar with modern methods of examining and treating the diseased organ. A number of technical manipulations are indicated in the treatment of these cases which can be performed only by an attendant who has had much experience and practice.

I believe, therefore, that in no other form of diseases is sanitarium treatment so necessary as in diseases of the stomach.

2. PHYSICAL METHODS OF TREATMENT.

Lavage of the Stomach.—For lavage of the stomach the same apparatus can be used as in aspiration of stomach-contents for diagnostic purposes—viz., the so-called stomach siphon, consisting of a soft elastic stomach-tube and a short glass tube connecting the stomach-tube with a long piece of rubber tubing into which a large glass funnel is inserted.

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[1] Fourth edition, edited by B. Fromm.

For therapeutic lavage of the stomach a stomach-tube with two lateral openings is to be preferred to one with a single opening at its lower end. I refer to page 67 for the technic of introducing the sound.

Certain authors—for instance, Penzoldt[1]—still recommend the employment of a guide, and claim that the introduction of the stomach-tube is easier if a so-called "mandrin" is used. I consider the mandrin superfluous, and maintain that it renders the introduction of the stomach-tube more difficult instead of making it easier. I have been in the habit of introducing the sound without a guide, and, to judge from my experience, this is by far the simpler and easier method.

As soon as the end of the stomach-tube enters the stomach, the funnel is slowly lowered; sometimes the stomach-contents will run out after this simple manœuver; if not, slight expression will cause it to flow. If nothing comes in this way, or if the flow stops after a part has run out, the funnel should be filled with lukewarm water (of about 88° F.), raised, and the water allowed to run into the stomach. As soon as the last portion of the water reaches the lower end of the funnel, the funnel is lowered and the water mixed with stomach-contents siphoned off.

I usually employ funnels holding about 500 c.c. of water; as a rule, I only allow one funnelful of water to run into the stomach and let the water run out again at once; I do this in order to avoid distending the stomach too much. This procedure is repeated until the wash-water is perfectly clear and contains no remnants of food. After the washing the patient should be instructed to express whatever water may have remained in the stomach. It is a good plan sometimes to instruct the patient to move his body to and fro in order that all the parts of the stomach-wall may be thoroughly washed by the water, or, as Fleiner[2] suggests, the patient may be instructed to lie down after the lavage itself was performed in the upright position; if this is done, remnants of food will frequently be found, even though the wash-water was perfectly clear while the patient was sitting up.

It frequently happens that a small piece of mucous membrane adheres to the opening of the sound when the sound is removed after lavage; accidents of this kind can usually be avoided if the tube is withdrawn while the funnel is raised, and not when it is lowered; in other words, while the water in the tube is still running into the stomach. This forces the mucous lining of the stomach away from the opening of the tube, whereas if the funnel is lowered, the mucous lining may be aspirated into the opening.

In washing out the stomach it is very important to determine with certainty that all the water that has been poured in has flowed out again; the best way to do this is to hold the rim of the funnel upward while the water is running out, and not downward, and not to evacuate the funnel until it is filled. In this way it can easily be determined whether or not the amount of fluid that flows out corresponds to the amount that was poured in.

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[1] Penzoldt, "Allgemeine Behandlung der Magen- und Darmkrankheiten," *Handbuch d. spec. Therap. innerer Krankheiten*, 1895, vol. iv.

[2] *Volkmann's Samml. klin. Vorträge*, new series, 1894, No. 103.

I would like to call attention to one other little point. Many persons make the following mistake in performing lavage of the stomach: they seem to be particularly anxious to empty the funnel completely and to get rid of the last drop of the mixture of water and food that flows out of the stomach. Some of them even attempt to cause all the water that is present in the tube to run out before they pour more water into the stomach. This plan is not a good one, for in the first place air may easily be forced into the stomach in this way and cause distention of the organ; in addition, the entrance of air may impede the outflow of water. In the second place they may aspirate the mucous lining of the stomach into one of the openings of the sound, and in this way injure the mucosa. The funnel should be emptied only so far that the new amount of water poured in rests upon the column of water; in this way the entrance of air into the stomach can be avoided.

Occasionally the outflow of water stops, notwithstanding all the precautions that may be taken. Very frequently this is caused by the entrance of coarse particles of food into the openings of the sound; in other cases the openings of the sound are no longer in the stomach-contents, either because the sound was not introduced far enough or because it became bent. In the latter case all that is necessary is to withdraw the sound a little; if this is done, the water will flow again. If, however, the sound is occluded, the attempt should be made to force the obturator out by pouring in more water. It will rarely be necessary to withdraw the sound, clean it, and reintroduce it. Sometimes the attempt may be made to remove the obstacle by forcing air into the tube from a blower; this manipulation is a simple one if a T-tube is inserted between the stomach-tube and the rubber tube leading to the funnel. The outer branch of this tube may be closed with a cork. If the sound becomes occluded, a rubber blower is attached to the outer limb of the T-tube, and the rubber tube leading to the funnel is at the same time compressed.

The arrangement of Friedlieb (see p. 72) is very useful in case an accident of this kind occurs. In this apparatus a small rubber bulb is inserted in place of an ordinary glass tube between the stomach-tube and the tube leading to the funnel; all that is needed is to compress the bulb, to close the tube leading to the funnel, and then to let the rubber bulb expand again. Another apparatus that is practical for this purpose is that designed by Strauss (see p. 73).

In the majority of cases the simple siphon is sufficient, and all aspirating apparatus are superfluous. Personally I have always gotten along very well without them.

The patient should never be allowed to perform lavage of the stomach himself; he may be allowed to introduce the stomach-tube, but in all subsequent manipulations he should have assistance. That part of the stomach-tube that protrudes from the mouth should always be held by the patient himself; this is a very necessary precaution, for otherwise the connection between the stomach-tube and the rubber tube leading to the funnel may suddenly become loosened and the tube glide into the stomach. This happened in a case of Leube's that we quoted above. It is well, therefore, that a second person should be present whenever lavage of the stomach is performed; this assistant should raise and lower the funnel, fill the funnel, etc. I never allow my patients to perform lavage of the stomach without the assistance of some second person; other physicians are in the habit of doing the same.

Many forms of apparatus have been devised for the purpose of enabling the patient to wash out his own stomach without assistance; I cannot recommend any of these contrivances, for many emergencies may arise in which the patient cannot help himself. Quite a number of these have been described.

The simplest method is to attach a glass triangle to the stomach-tube; the two limbs of the triangle that are not connected with the stomach-tube are attached to long rubber tubes; one of these rubber tubes leads to an irrigator that is suspended at moderate height; the other one leads to a vessel standing on the floor. If the lower tube is compressed and the upper one is allowed to remain open, the water flows from the irrigator into the stomach; if the upper tube is compressed and the lower one is allowed to remain open, the water mixed with stomach-contents will run out. It is simpler to use rubber tube clamps instead of compressing the tubes with the fingers. The irrigator should, of course, have a water-gauge, so that the patient can see how much water has flowed into the stomach. I also consider it essential that the vessel into which the wash-water runs should be graduated. This is not the case in the more common forms of apparatus. The only way, of course, to control the amount of water that runs in and the amount that runs out is to measure the quantities in both instances.

Litten[1] constructed an apparatus (see Fig. 15) that is similar to that described above, but simpler. It has the additional advantage of needing only one tube. The apparatus consists of an ordinary stomach-tube, rubber tube, and an irrigator[2] with a cock, also a middle piece made of hard rubber that is cylindric and perforated, and consists of two parts. About 80 cm. from the mouth the cylindric perforated middle piece (a), of hard rubber or glass, is inserted; this consists of two parts: the upper one (a_1) is conical and pointed, the lower one (a_2) is hollowed out to hold the upper one. As soon as water enough has run into the stomach, the cock (b) is closed, the middle piece disconnected, and the end (d) that is attached to the stomach-tube rapidly lowered. As soon as this is done the fluid will run from the stomach into a vessel that is held in readiness. If it is desired to allow more water to run in, the two pieces can be connected again and the cock (*b*) opened.

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[1] *Therapeut. Monatsh.*, 1893, p. 255.

[2] The irrigators in common use fail to effect a thorough cleansing of the stomach in most cases, as they do not hold a sufficiently large quantity of water. The necessity of repeatedly filling the irrigator with water alone makes it difficult for the patient to siphon out the contents of the stomach without assistance.

Formerly stomach-pumps were used, but nowadays they are no longer employed. We can get along very well without them, and their use should be condemned.

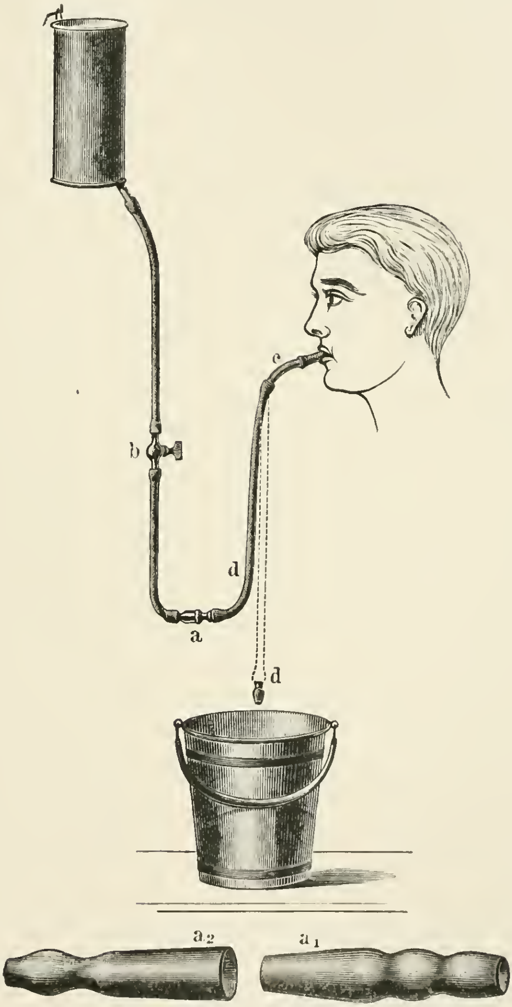

FIG. 15.—Litten's apparatus.

The time of day when the stomach should be washed out can be decided upon only after all the indications for therapeutic lavage of the stomach have been carefully considered.

In general we may say that lavage of the stomach is indicated in cases where the stomach cannot propel the ingesta into the intestine within the normal time, so that there is danger of abnormal decomposition and fermentation and of relaxation or overdistention of the stomach.

Lavage of the stomach is indicated, in the second place, if foreign products, like bile, mucus, etc., are present in the stomach and interfere with digestion or exercise some other unfavorable influence.

Gastrectasy proper and atony in which there is considerable stagnation of stomach-contents are the principal diseases of the stomach that call for lavage.

We distinguish two forms of ectasy—simple atonic ectasy and ectasy caused by stenosis of the pylorus or of neighboring parts. In both forms lavage is indicated, but in either form the effect of lavage will only be palliative, for washing out the stomach does not affect the disease itself. It cannot be stated definitely when lavage should be begun in atony; even in simple atony that has not yet led to ectasy I think that lavage is frequently indicated.

No one will deny that lavage of the stomach is clearly indicated in all cases of severe dilatation of the stomach in which the stomach-contents is decomposed or in which the stomach is unable completely to get rid of its contents overnight.

I believe that lavage of the stomach is indicated even if there is no ectasy in cases in which the stomach is unable to get rid of a carefully selected diet within the normal time; in other words, if, at the expiration of this normal time, large quantities of food are still found.

It has been argued that lavage, if performed in cases of simple atony, does not remove the *cause* of the disease; this is true, but the same applies to ectasy. We are, of course, unable to cure any chronic disease of the stomach by lavage alone; all we can do is to create favorable conditions that indirectly may assist in bringing about a cure.

Lavage is not indicated in every form of atony. In acute atony following the ingestion of improper food the stomach frequently gets rid of the decomposed material by vomiting.

Moderate degrees of atony that have existed for some time certainly often recover without lavage. If any one claims that in cases of this kind the stomach-walls are distended by the water that is introduced, this objection does not apply to lavage of the stomach, but to the person who performs it. It is self-evident that overdistention of the stomach should be avoided in performing lavage, and, as far as I can see, no one can deny that it is more rational to remove the decomposed and fermenting stomach-contents from an organ that is unable to get rid of it spontaneously than to allow the unhealthy mass to remain there.

If abnormal material is allowed to remain in the stomach, there is danger that the loss of tone will progress. It is impossible to determine in a general way when lavage should be begun; this matter must be decided differently in each individual case, and in order to arrive at a decision, all factors bearing on the question must be carefully considered.

It is altogether superfluous to enumerate the different diseases of the stomach in which lavage is indicated; besides, one form of stomach-disease may call for lavage in some instances and in others not. It is, of course, bad practice to neglect other methods for the sake of performing lavage; if there is a cancer with ectasy, lavage is indicated, but operative interference may be more urgently indicated than lavage. The same applies to stenotic ectasies following cicatricial ulceration. In cases in which the patient cannot make up his mind to an operation, lavage may be used as a palliative until the operation is performed.

Much has been written in regard to the time of day at which lavage may best be performed. Many authors, like Kussmaul, Fleiner, Boas, Wegele, recommend washing out the stomach before breakfast. They claim that if lavage is performed in the evening, a large proportion of the last meal will still be present in the stomach, and much nourishment will be withdrawn from the body. If lavage, on the other hand, is performed in the morning, the diseased organ will have had the advantage of the horizontal position occupied by the patient, and will have assimilated at least a portion of this material during the night. All this is undoubtedly true; the question arises, however, whether more of the food is really assimilated if it is allowed to remain in the stomach seven hours or twelve hours longer; it is doubtful to my mind whether it is better, in a case of atony of the stomach, to leave the abundant residue that is still present after seven hours in the stomach for another twelve hours,—in fact, to add a small supper,—or whether it is better to remove the decomposed non-digested and frequently fermented stomach-contents before introducing more food.

The best method of treating these diseases is not alone to force the stomach to assimilate the largest possible quantity of the food, but also to see that the ingesta do not remain in the stomach too long and do not damage the disordered organ. It might be argued that any lavage that brings up undigested food is a waste; even if lavage is performed in the morning and food is brought up, this is a waste, for the material siphoned out contains nutritive ingredients; these nutritive ingredients, it is true, might be digested by a healthy organ, but it is more than doubtful whether a diseased stomach can do anything with them, and, assuming even that a diseased stomach were capable of utilizing the nutritive material contained in this residue, the question still remains an open one whether it is beneficial or not to allow this material to remain.

I have been in the habit of teaching my pupils to distinguish two degrees of atony and of motor insufficiency; the one, in which the stomach is incapable of mastering a large midday meal in seven hours, but can very well get rid of a small supper in about ten to twelve hours, provided that the stomach was thoroughly cleansed before the administration of this supper; and the second form, in which the stomach cannot get rid of this small supper even in this long time. In the first form we speak of a mild, in the second form of a severe, degree of atony. If the stomach still contains considerable residue seven hours after the midday meal, it is atonic. A very convenient way in which to formulate a judgment in regard to the degree of atony is to wash out the stomach early in the morning, and to see whether food is still present in the organ; the stomach, of course, must not be washed out the evening before. It is well to wash out the stomach once in the evening after a test-meal; if large particles of food are found, the stomach should be thoroughly cleansed, a simple supper administered, and the organ washed out again the next morning; if it is found that the stomach is empty the next morning, it is demonstrated that the stomach was able to digest the smaller meal during the night; if, on the other hand, the stomach does not get rid of this small meal overnight, and if a considerable residue is found early in the morning, this demonstrates that the atony is more severe than in the other case.

I am in the habit of washing out the stomach in all cases in which considerable quantities of undigested food are found seven hours after a carefully selected midday meal; if I find that the stomach contains food in the morning after the organ had been washed out the evening before and a light supper administered, I am in the habit of washing out the organ again before breakfast. It is utterly useless to allow undigested food to remain in the stomach after the expiration of the normal time for digestion, for we cannot expect that even a portion of this residue will be assimilated. Assuming, for instance, that the secretion of hydrochloric acid is insufficient, all the albumin that is introduced will not be saturated with hydrochloric acid, and a portion of it will consequently remain undigested. It is certainly useless to expect the stomach to digest more albumin if it is incapable of digesting that which is still present in the stomach. It might possibly be argued that the small intestine would digest a portion of this residue; as a rule, however, the ingesta are more or less decomposed before they reach the intestine, so that there is even great danger of interfering with intestinal digestion. Under these circumstances it can hardly be expected that the residue of the food that is introduced on top of it will be digested in the stomach.

Assuming, on the other hand, that the opposite condition is present—namely, that we are dealing with a case of atony with hypersecretion; here, seven hours after a test-meal, an abundant fermenting liquid mass of amylaceous material will be found in the stomach. If this acid material is allowed to remain in the stomach and a supper forced on top of it, the stomach will become more and more distended, fermentation will continue to increase, and the new quantity of amylacea ingested will be saccharified like the older portions. All that is attained by postponing lavage of the stomach until the next morning is to favor the development of gas during the night, whereby the stomach is unnaturally distended and taxed during all this period; this cannot possibly aid digestion or improve the assimilation of the food. If, on the other hand, we remove the fermenting acid material in the evening before administering supper, the meat eaten will be well digested, and even a portion of the amylaceous material, if not given in too large quantities, may be assimilated in addition, the source of gaseous fermentation will be stopped, and the stomach will not be abnormally taxed nor abnormally distended.

In many cases it will be found that washing out the stomach before administering a carefully selected supper will leave the stomach empty in the morning, whereas if this preliminary lavage is omitted, the stomach will be found to contain numerous undigested particles of food in the morning. The effect of lavage in the evening is manifest; the stomach becomes capable of digesting a small supper, and the prolonged strain on the organ during the night is done away with.

If much food is found in the morning despite lavage in the evening, I also wash out the stomach in the morning. It is utterly useless to introduce more food into a stomach that already contains decomposed food. I consider it much more rational thoroughly to cleanse the organ before introducing more material; a portion of the new food will then be digested and will not at once begin to undergo abnormal fermentation.

The stomach is one of the organs whose function is periodic, consequently it needs intervals of rest. This applies to the healthy stomach, and, of course, with greater force to the stomach when it is diseased. One of the best ways to create favorable conditions for the cure of any disease of the stomach is to place the organ at rest for some time. Assuming even that we remove valuable nutritive material from the stomach by performing lavage in the evening, we are not certain whether or not the retention of food injures the diseased organ. We can, at all events, readily compensate any waste of food that may occur; that is simply a question of expense; as a matter of fact, we replace this loss at once by introducing new food into the stomach after it has been thoroughly cleansed. Under these conditions the food eaten is more thoroughly utilized than if it is given on top of old and partially fermented food.

I think the question whether or not the retention of old undigested food damages the atonic stomach can be answered in the affirmative. It is true that lavage of the stomach for the purpose of removing food that cannot be propelled into the intestine within the normal time is necessary only because we have no better means of remedying this defect; it would certainly be better if we possessed some means of forcing the stomach-contents into the intestine within the normal time-limit. Unfortunately, we do not possess any remedies that can do this, and so nothing remains but to remove all the food that has not left the stomach at the expiration of the normal time of digestion. This is absolutely necessary if we wish to avoid aggravating the atonic condition of the organ by continuously overtaxing the stomach. If we allow the food to remain in the organ longer than the normal time-limit, we allow the organ to become distended, and we permit irritation of the mucous lining. On the other hand, no advantage can possibly accrue, in the majority of cases, from the assimilation of nutritive material remaining in the stomach after the normal time for its digestion has expired.

I cannot agree with Boas when he says that another good argument in favor of performing morning lavage is that the stomach-contents in the morning furnishes many valuable diagnostic and prognostic clues; for, this author says, we can determine how great the reduction in normal peristalsis is at the time, and whether the treatment is increasing or decreasing gastric peristalsis. I believe that the stomach-contents in the evening gives us just as much information in this respect.

Although such experienced and excellent investigators as Kussmaul, Fleiner, and others recommend lavage in the morning, I am unable to agree with them, for the reasons mentioned above.

Minkowski and Quincke[1] occupy an intermediate position. Minkowski believes that if the patient is allowed to eat immediately after the morning lavage, the food introduced will immediately undergo fermentation, and in this way render the whole operation of lavage fruitless. For this reason he claims it is better to wash the stomach out in the evening, so that the organ may remain at rest during the night and the mechanical insufficiency be corrected as much as possible. Quincke, too, recommends washing out the stomach in the evening three hours after the last meal, and claims that the relaxed organ recovers more quickly if this is done.

I do not agree with Minkowski that food introduced immediately after lavage undergoes fermentation; on the other hand, I am very much gratified to learn that Minkowski, as well as Quincke, emphasizes the beneficial effects of lavage on the motor powers of the stomach that I have always emphasized. The advocates of morning lavage will again probably formulate the objection that by this operation valuable nutritive material is removed.

I fail to see any particular advantage in this method. If Minkowski fears the occurrence of fermentation in a stomach that has just been cleansed, he should also consider it dangerous to introduce a supper into an organ that still contains fermenting remnants of the midday meal and then to wash out the stomach late in the evening—that is, about three hours after supper. It appears to me that the patient would assimilate more nourishment if the stomach were washed out in the evening before supper. The wash-water might be medicated with antifermentative drugs, and then a small meal administered that the stomach is capable of digesting within a few hours. If under these conditions remnants of food or abundant quantities of gastric juice are still found the next morning, nothing remains but to wash out the organ again; in this way the most favorable conditions for the assimilation of breakfast are created.

The general practitioner will probably object to washing out the stomach of every case in which lavage of the stomach is indicated late at night—that is, three hours after the last meal. This question of convenience would, of course, be no objection to the method, provided it had other advantages. It has this advantage over morning lavage, that it rests the stomach and thus favors recovery; but it has the disadvantage, on the other hand, that it creates still less favorable conditions for the assimilation of food than the method I have recommended.

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[1] See "Berichte d. 67. Versamml. deutscher Naturforscher und Aerzte," abstract in *Centralbl. f. innere Med.*, 1895, No. 40.

My chief reason for entering so exhaustively into a discussion of these different methods is that they are of great practical significance in the treatment of diseases of the stomach.

It would be superfluous to enumerate all the different forms of stomach-disease in which therapeutic lavage is indicated; the physician must use his own judgment in each individual case, and must decide whether he considers lavage advisable or not. Washing the stomach for therapeutic purposes is contraindicated in all cases where aspiration of stomach-contents for diagnostic purposes is contraindicated. I consider gastric hemorrhage a contraindication, although Minkowski claims that it is not; irritation of the peritoneum also contraindicates lavage; certain diseases of the heart, as uncompensated valvular lesions, pronounced fatty degeneration of the heart, or wide-spread arteriosclerosis and similar conditions are considered by many to contraindicate lavage of the stomach; I think some clinicians go too far in this respect. The physician must strictly individualize in each case. If the patient is very weak, the sound should neither be used for diagnostic nor for therapeutic purposes.

Lukewarm water should be used for washing out the stomach; if it is desired to do more than simply to cleanse the stomach, the second and third washings may be medicated. It will depend on the nature of each case which remedies are to be added to the wash-water; the most common ones are alkalis or the saline constituents of Carlsbad, Wiesbaden, Vichy, Ems, etc., waters; in other cases certain antifermentative drugs, as salicylic acid, sodium salicylate, resorcin, sodium benzoate, boric acid, saccharin, creolin, lysol, and similar preparations may be added; sometimes astringents are useful, as nitrate of silver or subnitrate of bismuth, etc. We will discuss some of these remedies and the special indication for their employment in the following section on the treatment of stomach-diseases by douching; we will also discuss them again in detail in the sections on the different diseases of the stomach.

The Stomach-douche.—Malbranc,[1] in 1878, first described a procedure that he called internal stomach-douching. Kussmaul was the first to employ this measure in a case of severe gastralgia. Malbranc's description is as follows: Fill a glass funnel holding 300 gm., and connect it with the stomach-tube by a rubber tube; use a freshly prepared mixture of hot water and ordinary soda- or seltzer-water. The temperature of the mixture should be 100° F. The funnel is to be elevated to 1 m. or more above the cardia, and the fluid allowed to flow into the stomach through the tube; the funnel is then lowered while the patient is sitting up, and the water that has just run in is immediately allowed to run out again; later two or three funnelsful were poured in at once and allowed to run out after the water had remained in the stomach for some time; in this way, by raising and lowering the funnel, the stomach is douched with from two to three liters of the watery mixture at each sitting.

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[1] Malbranc, "Ueber Behandlung von Gastralgien mit der inneren Magendusche nebst Bemerkungen über die Technik der Sondirung des Magens," *Berlin. klin. Wochenschr.*, 1878, No. 4.

For a long time this method attracted little attention. In 1892 Rosenheim[1] again called attention to it, and reported his own experience. He did not, like Kussmaul, employ an ordinary stomach sound with large openings, but used a stomach sound that had numerous small openings (1 to 2 mm. in diameter) and one larger hole 3 to 4 mm. in diameter. If water is allowed to flow into the stomach through a sound of this kind, particularly if the funnel is held high enough to place the water under considerable pressure, all parts of the stomach mucosa are irrigated by the fine streams of water that gush out of the numerous little holes in the sound; no large stream of water, furthermore, violently squirts against any one place of the mucous lining. The chief advantage of the large opening in the axis of the sound is that the water can run off more quickly, and that, if necessary, certain solid morsels of food can be rapidly removed. The stomach douche should, of course, be employed only when the stomach is empty; and particularly in cases that are adapted to this method of treatment it is always possible that small particles of food may have remained in the stomach. The larger hole must, of course, not be too large, for otherwise the bulk of the water will flow into the stomach through this opening and no real irrigation of the whole mucous lining will be accomplished. Rosenheim advises irrigating early in the morning when the stomach is empty, or, if this is not practical, three to four hours after the first small breakfast.

Mild and slight degrees of motor insufficiency are particularly amenable to treatment by douching. According to Rosenheim,[2] the procedure can also be employed with advantage in mild degrees of chronic catarrh of the stomach with or without reduction in the motor powers, and, finally, in severe states of irritation affecting the sensory and the secretory apparatus. The author was not able, however, to determine that simple irrigation with water exercised any definite effect on the secretory function of the stomach; if he added salt, he saw an increase in the hydrochloric acid production; if he added silver nitrate, a considerable reduction in the secretion of gastric juice. Löwenthal[3] has also reported similar observations on the opposite effect of salt and nitrate of silver in irrigation of the stomach. At all events, I believe that these reports and my own experience justify the employment of a silver-nitrate douche in cases in which the stomach is very much irritated, particularly in its secretory and sensory functions.

The addition of chloroform-water is also useful in sensory irritation of the stomach.

Fleiner[4] has called attention to another effect of the stomach douche

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[1] *Therapeut. Monatsh.*, August, 1892.

[2] *Berlin. Klinik*, 1894, No. 71

[3] *Berlin. klin. Wochenschr.*, 1892, No. 49.

[4] *Samml. klin. Vorträge*, new series, No. 103.

—namely, the stimulation of the appetite. According to Fleiner, this effect can be increased in anorexia by adding bitters to the irrigating fluid. Kussmaul and Fleiner have found that infusions of hops or of quassia are particularly useful in this respect; other bitters and stomachics, particularly condurango and china bark, can also be used for the same purpose.

We see, therefore, that either warm water alone or medicated warm water can be used in the stomach douche. The chief remedies that have been used for medicating the wash-water are solutions of sodium chlorid (a teaspoonful to a liter), chloroform-water (50 to 60 gm. of chloroform shaken with 1 liter of water and then decantated), and silver nitrate solutions (1 : 1000).

The method of employing the stomach douche is a very simple one. If it is desired to use medicated douches, the stomach should first be thoroughly washed out several times with lukewarm water and the medicated fluid allowed to flow in afterward; the latter should remain in the stomach only a short time—half a minute to one minute—and then be allowed to run out, and the stomach subsequently washed out with lukewarm water.

Einhorn[1] has employed a method that is slightly different from the one described. He uses an ordinary spray apparatus to which is attached a soft Nélaton stomach-sound, some 70 cm. in length; within this tube is a second finer tube that is connected with the hard-rubber end of the capillary spray apparatus. As the spray is generated by an intimate mixture of the air that is driven out of the apparatus with the fluid, and as the fluid is forced out of the apparatus in a very fine suspension, it will reach every portion of the stomach-wall that the air reaches. One disadvantage of this method is that the stomach is dilated by the air that is forced into it.

Einhorn claims that his method is useful—first, for disinfecting the stomach mucosa; second, for producing an astringent effect; third, for causing local analgesia in gastralgia. The spray apparatus should, of course, be used only when the stomach is empty.

Massage of the Stomach.—The stomach being a muscular viscus, it seemed natural to use massage as in any other muscular organ; it seemed indicated particularly where it was desired to raise the tone of the relaxed muscularis. Another indication for massage appeared to exist in gastrectasy with a great reduction of the motor power of the stomach, for it was expected that by suitable manipulation the propulsion of the ingesta from the stomach into the intestine might be accelerated.

It appears that in both these conditions massage is a useful procedure. At the same time it is impossible to render final judgment in regard to its value, as enough has not yet been published on the results obtained by this method.

The most suitable lesion of the stomach in which massage is indicated is undoubtedly simple atony. If atony is complicated with ulcer, or if there is recent inflammation of the stomach, massage is not indicated. In ectasy the procedure may also be tried, particularly in the simple atonic form, not so much in that form that is due to stenosis of the pylorus. If ulceration or irritation of the stomach is present, massage is, of course, again contraindicated.

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[1] *New-Yorker med. Wochenschr.*, October, 1891.

If there is great accumulation of gas in the stomach, massage should not be performed; it is more rational in these cases to suppress fermentation by methodic washing of the stomach, by the internal administration of antifermentative drugs, and by a suitable diet. Cséri,[1] however, has shown that it is possible to remove the gases that accumulate in the stomach by massage. This procedure should be employed only for the temporary removal of large amounts of gas, not for the methodic treatment of gaseous fermentation; because, of course, massage may possibly remove whatever gas happens to be present, but certainly cannot prevent its reformation.

Other conditions in which massage has been advised by different authors are cases of reduced secretion of gastric juice, certain forms of nervous dyspepsia and of gastroptosis. I hardly believe that much benefit can be derived from massage in these conditions.

The chief sphere of usefulness for massage is the different forms of atony and of atonic ectasy. Zabludowski[2] has shown that good results may occasionally be obtained even in cases of ectasy that are due to stenosis of the pylorus. I have personally seen good results in many forms of ectasy, and have repeatedly succeeded in improving the motor powers of the stomach and in causing a more rapid evacuation of the stomach-contents by a regular system of massage; in some cases massage did not benefit. Cséri[3] is not so favorably impressed with this method, for he says it is only exceptionally possible to force stomach-contents into the intestine through the pylorus.

Different authorities do not agree in regard to the best time for performing massage of the stomach. In cases in which the main object of the manipulation is to force the stomach-contents into the intestine massage must be performed when the stomach is full; in cases of this kind it appears to me the best time is late—that is, six hours after the chief meal—so that the stomach may have plenty of time to convert the food into a fine mass, and in this way facilitate its passage through the pylorus.

In cases, on the other hand, in which massage is intended to strengthen the stomach, to improve the tone of its muscularis, as in simple atony and in mild degrees of atonic ectasy, massage should be performed several hours earlier.

Cséri advises massaging two to three hours after the meal; other authors advise performing massage early in the morning when the stomach is empty. An objection to the latter plan is that the stomach, when it is empty,—that is, contains neither food nor air,—is contracted and cannot be palpated, so that under these circumstances it appears almost impossible really to massage the stomach. I do not think that any general rules in regard to the time of performing massage of the stomach can be formulated; much will depend on the degree of atony and motor insufficiency.

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[1] *Wien. med. Wochenschr.*, 1894, Nos. 46–48.

[2] *Berlin. klin. Wochenschr.*, 1886, Nos. 26–28.

[3] *Loc. cit.*

The technic of performing massage of the stomach is the following: Massage should be performed daily: in the beginning, for a short time only—that is, for three or four minutes; later, for from five to ten minutes. The patient should be placed in a horizontal position, the legs flexed at the knees and the hips in order to relax the abdominal muscles. According to Cséri, the masseur should place his left hand on the right hypochondrium and should perform stroking movements with the right hand from the fundus toward the pylorus; the fingers should be stretched, and the thumb should also be extended as far as possible. In this way it aids the action of the fingers. While the right hand is performing stroking motions from left to right, the left hand performs counterpressure, so that the stomach is held between both hands. In cases of dilatation and dislocation of the stomach the direction of the stroking must be adapted to the position of the organ in each individual case. Later, the region of the stomach is kneaded; the kneading and stroking motions should alternate.

Zabludowski uses another method that is different from the one described, and is based on the personal experience of this author in the treatment of ectasy due to stenosis of the pylorus. He advises picking up a large fold, consisting of abdominal wall, stomach-wall, and the contents of the stomach, between the thumb and the four fingers of the right hand. Rapid propulsive motions are now executed, and the attempt is made to throw the contents of the stomach against the pylorus. I do not believe that this manœuver will succeed in the majority of cases; it seems possible only when the abdominal walls are very much relaxed.

According to Zabludowski, it is possible, in cases of ectasy of high degree with hypertrophy of the stomach-wall, to divide the stomach into two portions by pressing downward with the finger-tips against the spinal column: the smaller portion will be the one near the esophagus; the larger one, near the pylorus.

The material inclosed in the latter pocket acts as a bougie, and if pressure is exercised on this part of the stomach, the pylorus will be dilated by this bougie. The manipulation consists in performing rapid vibratory movements and in exercising deep pressure transversely across the abdomen. Occasionally, Crédé's method may be employed; this author also manipulates the abdomen transversely, and in this way forces the food toward the pylorus. Zabludowski, however, does not recommend stroking (effleurage) nor tapping (tapotement). According to this author, the whole manipulation should not consume more than two or three minutes, and the whole sitting should last only about ten minutes. Massage of the abdomen should never be painful, not even disagreeable, to the patient. Zabludowski, in addition, recommends placing the patient on his back during one-half of the time; on his right side during the other half. In cases in which the stomach performs active peristaltic movements spontaneously or in which the organ is very hard, massage is not indicated.

Massage of the intestine, according to Ziemssen[1] and Wegele,[2] can follow massage of the stomach in cases of atony of the intestine and of chronic constipation combined with gastrectasy.

Massage should, of course, be performed by the physician himself. Occasionally it may be permissible to allow the patient to massage himself. I have taken care of a number of my colleagues who learned to perform massage on themselves; these were all cases of motor insufficiency of high degree. Self-massage is performed either with the hand or with a wet cloth that is folded many times; the patient can use the latter to perform stroking motions from left to right. It is, of course, preferable to have a physician who is familiar with the technic of stomach massage perform the manipulations.

Electric Treatment.—Treatment of diseases of the stomach by electricity, like massage of the stomach, is not generally employed; it appears, however, to judge from clinical experience, that the electric current can exercise a marked influence on the functions of the stomach.

Physiologic experiments that have been carried on in this direction have yielded certain results; in regard to some of the functions of the stomach they are, however, altogether negative; in other instances they do not correspond to the effects noticed in human subjects; in fact, seem to be altogether contradictory. Certain observations published on the effects, for instance, of the electric current on the secretion of gastric juice have been reported by Ziemssen[3] and Rossi;[4] it was found by them that the electric current is, in fact, capable of stimulating the secretion of gastric juice; Hoffmann,[5] in my clinic, performed similar experiments on human subjects and demonstrated that the secretory apparatus of the stomach can be influenced by the electric (galvanic) current. The test was carried out as follows: Two large plate electrodes were employed, and a strong current passed; each sitting lasted for twenty minutes; the patients were treated on alternate days, and the secretion of the stomach examined while the organ was empty. It was found that on the days of treatment a considerable quantity of gastric juice was present in the stomach, and that on the days between treatments this was not the case.

These results seemed to indicate that it was possible to influence the secretion of gastric juice by galvanic electricity; they did not, however, explain how the nerves of the stomach were influenced by this treatment. It seemed probable that the gastric branches of the vagi were directly or indirectly stimulated; that the vagi contain secretory fibers is made probable by a number of observations, notably those of Beynard and Loye,[6] who succeeded in causing a secretion of gastric juice by simple stimulation of the vagi forty-five minutes after the death of a criminal who had suffered capital punishment. Experiments by Pawlow and Schumowa[1] on dogs also showed that the vagi are concerned in gastric secretion; the dogs that these investigators used had both a blind esophageal and a stomach fistula. Everything that the animals ate was immediately passed through the esophageal fistula; notwithstanding this, an abundant quantity of gastric juice was seen to flow from the fistula as soon as the dogs were fed; if the vagi were severed, this secretion of gastric juice did not occur. Separation of the splanchnics did not influence the secretion.

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[1] *Klin. Vorträge*, 1888, No. 12.

[2] Wegele, *Die atonische Magenerweiterung und ihre Behandlung*, Munich, 1894.

[3] *Klin. Vorträge*, No. 12; *Die Elektricität in der Medicin*, 1887.

[4] *Lo Sperim.*, 1881.

[5] *Berlin. klin. Wochenschr.*, 1889, Nos. 12, 13.

[6] *Progrès médicale*, 1885, No. 29.

The exact manner in which gastric secretion is influenced by electricity remains more or less obscure. The fact that this can occur seems established. Of late, however, these apparently established results have been questioned. Goldschmidt,[2] for instance, failed to cause any alterations in the secretion of gastric juice in human subjects either by gastrofaradization or by gastrogalvanization.

I am unable to explain these contradictory results. Goldschmidt expresses the opinion that the subjects that we experimented on had higher acidity; this, however, is not the case, at least not in the majority of our cases, and even if it were so, it would not explain the peculiar discrepancy.

In regard, however, to other effects of the electric current it is well established that it can affect the motor powers of the stomach and the sensibility of the organ. This action of the current has never been demonstrated by direct experimentation, but has been deduced from clinical experience. All the physiologic experiments that have so far been performed in this direction seem, on the contrary, to furnish negative results. Goldschmidt, for instance, failed to see any increase of the motor powers of the stomach in normal subjects that were treated with electric currents; the same negative result was seen in 2 cases with dilatation of the stomach and reduced motor power. Meltzer[3] has recently reported a number of animal experiments, and also chronicles negative results. Meltzer passed very strong induced currents through the fundus of the stomach after exposing the organ, and never noticed any contractions of the muscularis. The only portion of the stomach that seemed to contract was the right side, and it seemed as if the contractions grew stronger as the electrode approached the pylorus. He never succeeded, however, in causing any contractions of the pylorus by irritating the corresponding portion of the mucosa. If the stomach was exposed and one electrode placed on the abdominal wall near the stomach, the other one applied to the back or introduced into the stomach, a strong current caused contraction of the abdominal muscles, but never of the stomach.

These results were all seen in small animals, and they can probably not be directly applied to human beings, particularly not to subjects with diseases of the stomach. Hirsch[4] has shown that narcosis alone may stop movements of the stomach that were very pronounced before the administration of the anesthetic. Laparotomy itself frequently influences the movements of the intestine. For these reasons more importance should be attached to physiologic experiments in human beings, but these, as we have seen, also yield negative results; for the present, clinical experience and physiologic experimentation do not agree. From the point of view of the general practitioner, however, clinical observation is undoubtedly the more important of the two.

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[1] *Centralbl. f. Physiol.*, vol. iii., No. 6, p. 113.

[2] *Deutsch. Arch. f. klin. Med.*, vol. lvi.

[3] *Centralbl. f. Physiol.*, 1895, No. 8.

[4] *Centralbl. f. klin. Med.*, 1892, No. 47.

It was believed for a long time that the electric current could increase absorption in the stomach. The experiments that have been performed in this direction are not conclusive, in part because the method that was employed (the salol method) is uncertain, in part because the differences obtained, as, for instance, in Einhorn's[1] recent experiments, are so small that they remain within physiologic fluctuations.

In contradistinction to these rather unsatisfactory physiologic results, the therapeutic results obtained from the application of the electric current are uniformly favorable.

Even Goldschmidt, who, as we have seen, obtained altogether negative results in his physiologic experiments on man, says that his therapeutic experience indicates that direct electrization of the stomach is an excellent means for treating nervous disorders of the stomach, and is also of value in treating organic lesions of the viscus.

There is a great diversity of opinion in regard to the best method of applying the electric current; some prefer intraventricular, others extraventricular, application. There is also a diversity of opinion in regard to the kind of current to use. Most observers, however, prefer the faradic current when the motor power is reduced, and the galvanic current when there are symptoms of sensory irritability.

The effect of the two kinds of current on gastric secretion has been studied by many authors. The results reported are not uniform. Einhorn,[2] for instance, arrived at the conclusion that the faradic current increases secretion and the galvanic current decreases it. Hoffmann, on the other hand, claims that the galvanic current improves secretion. Bocci[3] succeeded in causing increased contractions of the stomach and an increase in the secretion of gastric juice by intraventricular faradization in animals, whereas Goldschmidt failed to influence gastric secretion either by faradization or by galvanization of the stomach.

There is no agreement in regard to the advantages of intraventricular and extraventricular application of the current. From a therapeutic point of view the intraventricular method seems preferable, because the diseased organ is treated directly. As against this we must remember Meltzer's statement that he never succeeded in causing contractions of the stomach by irritating the mucous membrane. In practice most physicians prefer the percutaneous application of the current, because it is simpler and more easily executed than intraventricular electrization.

Still other objections have been formulated against intraventricular application of the current. Von Ziemssen,[4] for instance, has called attention to the fact that a stomach-tube will always turn to the left when it enters the stomach, owing to the normal rotation of the axis of the esophagus to the left, and that for this reason the electric current will always be applied principally to the same point of the greater curvature. This point, as Meltzer has shown, cannot be forced to contract even by very strong electric currents. The results that von Ziemssen obtained from the application of electric currents, particularly in ectasy of the stomach, are altogether unsatisfactory, for the procedure was very exhausting to the patient and the effect hardly appreciable. Von Ziemssen has, therefore, abandoned the intragastric application of the electric current, and recommends only percutaneous application.

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[1] *Zeitschr. f. klin. Med.*, vol. xxiii.

[2] *Deutsch. med. Wochenschr.*, 1893, and *Zeitschr. f. klin. Med.*, vol. xxiii.

[3] *Lo Sperim.*, Giugno, 1881.

[4] *Klin. Vorträge*, 1888, No. 12.

The best technic in percutaneous electrization is the one recommended by von Ziemssen. This author employs plate-electrodes of 600 to 500 c.c. in diameter; the larger of the two is placed upon the anterior abdominal wall, in the region between the pylorus to the fundus; the smaller one, from the fundus to the spinal column, corresponding to the position of the stomach. The electrode should be thoroughly moistened. The distance between the two electrodes should not be more than one or two centimeters. The strength of the current must be very great if such large electrodes are employed. If the induced current is employed, or if the constant current is commuted, strong contractions of the abdominal muscles, etc., should occur; this shortening of the abdominal muscles and of the muscles of the back causes movements of the body and contractions of the diaphragm. These are not painful, however (von Ziemssen).

Kussmaul[1] calls attention to the fact that possibly the favorable effects seen from percutaneous electrization are not due to any direct influence on the musculature of the stomach, but are due to the contractions of the abdominal muscles. Kussmaul, by the way, was the first to advise the internal application of the electric current.

Meltzer has published a number of investigations that seem to support Kussmaul's view.

A number of instruments have been recommended for the intragastric application of the electric current. Kussmaul was the first to introduce a stomach sound with a copper wire into the stomach. The same author was the first to report favorable results from the application of the faradic current with this instrument in a few cases of dilatation of the stomach. Bardet[2] constructed another electrode that was an improvement on Kussmaul's, for with this instrument it was possible to apply galvanic electricity to the interior of the organ. The electrode in this apparatus did not come in contact with the stomach-wall, but with a column of water that was first poured into the stomach; in this way erosion of the stomach mucosa was avoided.

Von Ziemssen employed a similar apparatus; he used a stomach sound that could be employed for washing out the stomach and for electrizing the organ at the same time. In this instrument the metallic head of the mandrin was fixed in the sound, and contact with the stomach-wall was produced through the water that was allowed to run into the stomach before the current was turned on. The circuit was closed by applying the large electrode to the anterior abdominal wall.

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[1] *Arch. f. Psychiatrie und Nervenkrankheiten*, 1878, p. 205.

[2] *Bull. gén. de thérap.*, 1884.

Einhorn[1] has constructed a peculiar instrument for the same purpose. He uses an electrode that can be swallowed. The apparatus is shaped like a small egg, and consists of a hard-rubber shell that is pierced by a number of small holes about as large as pin-heads; within this shell is a ball of copper or brass. The patient swallows this egg-shaped apparatus, which is connected with a thin wire inclosed within a thin rubber tube. The thin wire frequently catches in the esophagus, and Ewald,[2] in order to remedy this defect, has constructed another apparatus in which Einhorn's egg-shaped electrode is used. He uses a very thick rubber tube to cover the wire, the walls of which are about 1½ mm. thick. This rubber tube can easily be pushed into the stomach and does not distress the patient.

Rosenheim,[3] like Ewald, uses a thin stomach-tube. Within this the wires pass downward to an oval button that has several perforations; the end of the electrode is attached to a button in such a way that it does not come in immediate contact with the stomach-wall. Before the apparatus is introduced, the patient's stomach should be washed out and one or two glasses of water given; if the stomach is filled with water in this way, the current is more evenly distributed throughout the organ. The second electrode is either applied in the epigastric region or in the back; it is also possible to introduce one electrode into the stomach and another one into the rectum, so that the whole intestine is electrized. Ewald reports some very surprising results with this method in cases of intestinal atony combined with slight degrees of dilatation of the stomach. In other cases that he reports he saw no good effects from this method.

Wegele[4] has recently described another electrode that seems to me to be an improvement upon the ones just described. This instrument consists of a metal spiral that is so soft that it follows every movement of the rubber tube. The spiral can readily be inserted into any stomach sound, and the patient, when he swallows the sound, does not notice the presence of the metallic coil. The flexibility of the spiral is due to the large number of turns that the thin metal wire makes; there are two turns for each millimeter, so that there are about 1500 turns in a spiral of 75 cm. At the upper end of this coil of wire is a metallic tube some 5 cm. thick, to which is attached a contact screw for attaching a wire connected with one pole of the electric battery. This tube is inserted between the stomach sound and the funnel tube in place of the ordinary glass tube. The chief advantage of this apparatus is that it can be used for lavage of the stomach, electrization, and siphonage of the water that is poured in, and that it is not necessary to remove the sound for each of these manipulations. At all events, it can be said that this electrode is particularly adapted for general use because its introduction is no more difficult than that of an ordinary soft stomach-tube.

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[1] *Deutsch. med. Wochenschr.*, 1883, and *Berlin. klin. Wochenschr.*, 1881.

[2] *Klinik der Verdauungskrankheiten.*

[3] *Berlin. Klinik*, No. 71.

[4] *Therapeut. Monatsh.*, April, 1895.

It appears to me, therefore, that Wegele's apparatus is the best of all the stomach electrodes that have so far been described.

Different authors seem to disagree in regard to the indications that exist for the application of galvanic or faradic electricity. Einhorn[1] has performed the most numerous and the most thorough investigations into direct electrization of the stomach, and this author recommends direct gastrofaradization chiefly in cases of dilatation of the stomach that are not due to stenosis of the pylorus, but to relaxation of the stomach musculature, whether or not this dilatation is accompanied by hyperacidity or a subnormal degree of acidity.

Cases of relaxation of the cardia and of the pylorus are favorably influenced by faradization. Gastrogalvanization, on the other hand, has been successfully employed by Einhorn in many cases of obstinate gastralgia.

Rosenheim, in opposition to Einhorn, is inclined to employ galvanization instead of faradization in cases of motor insufficiency of the stomach. This investigator expresses the opinion that disturbances in the sensory sphere of the stomach are most adapted to electric treatment, and that in these conditions the galvanic current is to be preferred to the faradic. Rosenheim does not use electricity in secretory anomalies of the stomach, because he claims never to have seen any favorable results from this method of treatment. He believes that medicated douches or drug treatment alone will accomplish much more in these conditions.

Brock[2] also emphasizes the favorable effect obtained from direct galvanization of the stomach in neuroses of the organ, although he is not inclined to acknowledge the efficacy of this method as unreservedly as Einhorn.

According to Goldschmidt,[3] there is no great difference between the effect of endogalvanization and endofaradization. He claims, however, that endogalvanization (with the anode in the stomach) is more adapted to painful disturbances of the organ, and that endofaradization is better in functional disorders of the organ.

Von Ziemssen, who, as we have already said, prefers percutaneous application to intraventricular application (in contradistinction to Einhorn, Ewald, Rosenheim, Brock, and others), claims that the most important effect of the electric current on the stomach is the stimulation of the appetite, particularly in cases with nervous anorexia, and that this improvement of the appetite is accompanied by an increase in the active powers of digestion.

He claims that persons with nervous anorexia are frequently able to eat with good appetite and to digest their food comparatively well after electric treatment. Ewald also calls attention to the benefits of electricity in cases of nervous anorexia.

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[1] *Zeitschr. f. klin. Med.*, vol. xxiii

[2] *Therapeut. Monatsh.*, June, 1895.

[3] *Loc. cit.*

According to von Ziemssen, the majority of cases that are suitable for electric treatment of the stomach at the same time call for some stimulation of their general nervous energy. He recommends the application of dry brush electrization of the skin of the abdomen, the back, and the chest, to be applied after the stomach treatment. He advises giving this cutaneous electrization for from two to three minutes, and to use large brushes—about as large as an adult hand—and to employ a very strong induced current.

Von Ziemssen claims that the following diseases are most suitable for treatment by electrization : nervous or neurasthenic dyspepsia, atonic reduction of the powers of digestion, habitual vomiting, and the different paresthesias of nervous or mechanical origin ; further, irritative conditions of the sensory and secretory fibers of the stomach-wall that are due to mechanical conditions, and finally atonic ectasy—not, however, large ectasies that are due to stenosis of the pylorus.

Whereas a great diversity of opinion still exists in regard to some of the points under discussion, most authors agree that the neuroses of the stomach, particularly the sensory neuroses, are most suitable for electric treatment. The chief diseases that belong to this category are nervous anorexia, habitual nervous vomiting, and other nervous disturbances of the stomach. In cases of this character the galvanic current is unquestionably to be preferred to the faradic, and direct galvanization is better than percutaneous galvanization, although in cases in which intraventricular electrodes cannot be obtained, the percutaneous method may be employed.

The galvanic current can also be used to advantage in attacks of pain and other symptoms of irritation following, for instance, adhesive perigastritis and similar conditions.

The electric current is secondly useful in cases of simple atony and atonic ectasy, but not in cases of ectasy that are due to stenosis of the pylorus. In the first form of ectasy it is, of course, necessary to continue the use of other valuable adjuvants to the treatment, as lavage of the stomach, massage, and similar manipulations. In this group of diseases the faradic current is preferable to the galvanic current, and the percutaneous method is probably better than the intraventricular mode of application. The galvanic current may finally be tried in certain anomalies of gastric secretion, provided they are not of nervous origin.

As far as I can see, these are the only diseases in which electric treatment is so far indicated. It will require more experience than we command to-day to formulate more precise indications for this treatment. The general practitioner in the majority of cases will, as a rule, be able to get along very well without electricity ; at all events, he will be able to treat his patient with other methods that are just as effective. The chief spheres for electric treatment of the stomach are the nervous disorders of the organ, and cases of this kind should preferably be treated in sanitaria.

[The use of electricity has followed a somewhat different course in

America than in Europe. In 1887 the writer began the use of this method of treatment, and used for its application a combined electrode and stomach-tube, which was described in 1889[1] and again in 1892.[2] This instrument closely resembles that which has recently been used and described by Wegele. In 1891 Einhorn described his deglutable stomach electrode, and since then, by means of one or the other of these instruments, the treatment of stomach-diseases by electricity has been largely practised here. To us there seems to be little benefit in the percutaneous application of electricity as practised by von Ziemssen. However, excellent results have been obtained by the intragastric method; the objections raised by von Ziemssen to the employment of the intragastric electrode are not borne out by facts. There is no more objection to intragastric electric treatment than to the use of the stomach-tube. It has been found that in the treatment of a number of affections the method produces better results than any other. Among these affections may be mentioned atony, gastrectasia resulting from atony, hypochlorhydria, paresthesia, hyperesthesia, anorexia, and gastralgia. A good many years' experience teaches that the benefits obtained are not imaginary and are generally immediate. The stomach should be emptied before beginning the treatment, and this, among other reasons, leads one to favor the combined stomach-tube and electrode. As to the nature of the current selected, it may be said that the faradic has been most used, is perfectly safe, and in some conditions is preferable. That it is competent to produce a free secretion of active gastric juice there can be no doubt, as has frequently been shown both experimentally and clinically. It should be said that the continuous current is quite as effectual in this respect. As to the stimulation of motion by the local application of electricity, the clinical and experimental results are in some respects contradictory. The accumulation of cases of atony and diminished motility that have been relieved by electric treatment is convincing evidence of the utility of the practice. Nevertheless, I have shown experimentally[3] that aside from producing prompt pyloric contraction, there was little motion excited in the gastric walls of dogs, even when very powerful currents were employed. Meltzer confirmed this in 1895. Nevertheless, the fact remains that motor activity is improved by electric treatment, and we agree with Einhorn that it would be inconvenient to get along without its assistance.—Ed.]

Hydriatic and Orthopedic Methods.—Hydrotherapeutic treatment is frequently indicated in certain diseases of the stomach. We must distinguish between general hydrotherapy, in which the whole body is rubbed down, in which full baths or packs, general douches, and similar methods are employed, and local hydrotherapy. The first-named methods are intended to exercise a general effect on the patient, either to quiet him or to refresh or stimulate the whole nervous system. It is claimed that the functions of the stomach are indirectly and secondarily influenced in this way. These general procedures are indicated chiefly in functional disorders of the stomach, particularly in the different forms of so-called nervous dyspepsia; they are less useful in organic diseases of the stomach proper.

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[1] *Med. Record*, November 9, 1889.

[2] *New York Med. Jour.*, July 30, 1892.

[3] *New York State Society*, reported in 1892.

It would lead us too far were we to discuss the details of these methods in this place. More will depend on the general condition of the patient, the state of the whole nervous system, than on the local disturbance. In some cases wet rubs, full baths, general douches (in some cases with carbonated waters or waters that are medicated in some other way), or general packing, etc., must be employed.

If it is desired to give the patient the benefit of general hydriatic treatment, he should be sent to a cold-water cure. Procedures of this kind cannot be successfully employed at the home of the patient.

Local hydriatic measures are very important. They are applicable to a great variety of diseases of the stomach—both to organic lesions and to nervous disturbances of the organ.

Cold may be employed either in the form of cold or frozen compresses or in the form of small ice-bags. Leiter's coils are very useful; they consist of tubes through which a stream of cold water is allowed to flow continuously. Cold applied locally to the region of the stomach is useful chiefly in gastric hemorrhages and in periodic irritation of the stomach.

A very popular hydrotherapeutic procedure is the application of so-called Priessnitz compresses. These are made by dipping a towel that is folded several times into cold or warm water, wringing the towel out until no more water can be expressed, and then placing it on the stomach region. The towel is covered with a layer of flannel, or, better, with a layer of gutta-percha paper or oiled silk. The whole is then fixed in its place by a broad flannel binder. The temperature of the water is usually from 55° to 77° F. The temperature of the water is selected to suit the sensations of the patient. If it is desired to cause much irritation, the water should be used colder; if the irritation is intended to be only slight, the water should be warmer.

These Priessnitz compresses are used to great advantage in almost all painful diseases of the stomach. As a rule, it is sufficient to change the compresses twice a day if care is taken that evaporation is prevented by some suitable covering. It is not definitely determined how this application stops pain and quiets the patient; as a matter of fact, however, the majority of patients feel very much relieved after the application of a Priessnitz compress, and the pain seems to disappear. This may explain why some patients claim that an application of this character puts them to sleep.

In other cases, particularly in violent cardialgia or in other very painful diseases of the stomach, hot applications, particularly in the form of cataplasms, are indicated. Leube was the first to recommend these applications in the treatment of round ulcer of the stomach. Cataplasms can be made from linseed or linseed and bran, boiled with water to form a hot poultice. If necessary, bread and milk can be used for the same purpose. The pultaceous mass is wrapped in a cloth and applied as hot as the patient can bear it. As soon as the poultice begins to cool off, it is replaced by a new one. It is very convenient to use one of the apparatus that are on the market for keeping the cataplasms at a constant temperature ; or so-called constant cataplasms may be employed. These are boiled in water for about twenty minutes, and then wrapped in a cloth. So-called "Filzschwamm" (felt-sponge) may be used in place of cataplasms, as this material is more easily applied. All that is needed is to dip it into boiling water, to squeeze it out, and to cover it with some impermeable material.

Dry heat is used less frequently in diseases of the stomach than moist heat; it is indicated particularly in sudden spasmodic affections of the stomach. Here hot cloths or hot plates that are wrapped in cloths may be employed. So-called "warm boxes" that have recently been imported from Japan, consisting of a metal box covered with cloth, are useful. Glowing punk is placed into the metal box, and the apparatus kept warm for a long time in this way. In general, however, moist warm compresses are preferable to dry heat.

I have had no personal experience with the method that Winternitz[1] recommends. He prescribes the application of a cold wet compress that is wrapped in a dry cloth ; over the latter pass several coils of a rubber tube through which circulates water of 104° F. Winternitz claims great advantages from this method, not only in neuroses of the stomach, but also in organic diseases of the organ. Strasser,[2] from his personal experience in Winternitz's institute, claims that this method is very useful in all catarrhal affections of the stomach, and that it is invaluable in hyperesthesia of the organ and in pathologically increased irritability both of the sensory and the motor nerve-endings.

Strasser says that it is surprising to see patients who had been unable to take food for months or years without pain or a tendency to vomit and who had been in the habit of vomiting their food for a long time, enjoy a meal and retain the food without pain and without nausea after a single application of Winternitz's method.

Other hydriatic measures that merit mention are local douches, so-called fan douches, and Scotch douches. When these different douches are employed, both thermic and mechanical effects are seen. In the so-called Scotch douche the general effect of the treatment is increased by the sudden changes of temperature. The Scotch douche is applied by allowing a stream of water to play on the region of the stomach. The temperature of the water is changed about every twenty seconds (95° and 55° F.). The whole treatment lasts for about two or three minutes. Rosenthal[3] praises the Scotch douche and claims that it is a splendid method for stimulating the energy of the gastric musculature. Von Ziemssen claims the same.

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[1] *Deutsch. Medicinal-Zeitung*, 1891.

[2] *Blätter für klinische Hydrotherapie und verwandte Heilmethoden*, 1895, vol. v., No. 5.

[3] *Magenneurosen und Magenkatarrh, sowie deren Behandlung*, Vienna, 1886.

Sitz-baths are rarely used by patients with diseases of the stomach —at least not for the treatment of the stomach trouble.

I cannot enter into the details of general hydriatic measures nor the indications for their use. They are employed less frequently for the local disturbances than for the general conditions of the patient, particularly the nervous disorders that he may be suffering from, so that the indications for this method of treatment are identical with the indications for the hydriatic treatment of neurasthenia. I have already mentioned that hydriatic treatment of this kind can be carried on only in some institute.

Orthopedic methods of treatment are intended to give the stomach support in different diseases of the organ. They are applicable to cases of relaxed abdominal walls, of pendulous abdomen, of diastasis of the rectal muscles complicated by ptosis and dislocation of the stomach. Here some appropriate form of bandage or abdominal binder should be applied in order to support the abdominal walls, and in this way enable the stomach to remain in its normal position. If the organ is already very low down, a bandage of this kind will keep it from dropping further. This bandage is also useful in cases of ectasy; it cannot aid in reducing the size of the organ nor in reëstablishing its tone, but it gives the atonic and distended stomach a fixed point of support.

A simple flannel abdominal binder is, of course, not sufficient to accomplish this. It is necessary to use apparatus that can either compensate the loss of tone of the abdominal walls and actually constitute, as Landau says, artificial abdominal walls, or that are capable of exercising pressure in some one direction. The apparatus must be modified to suit the peculiarities of each case, and one apparatus will be used if it is desired to compensate the lost tone of the abdominal walls, and another if it is desired to create a support in some one localized area, the exact location of which will depend on the position and the degree of distention of the stomach. There is no bandage that is universally applicable to all cases; we are not able to say, therefore, that this, that, or the other form of bandage is suitable for any definite case; we can only say that in dislocation of certain other organs, as, for instance, of the kidney, but not of the stomach, as there are too many modifications. If the whole trouble is due to great relaxation of the abdominal walls, a bandage should be employed that supports the abdominal walls *in toto*, or, as Landau puts it, artificial abdominal walls should be supplied. Landau has constructed an abdominal corset that is suitable for this purpose. Bardenheuer has also constructed a very useful apparatus of this kind; it is particularly applicable to cases of pendulous abdomen in obese subjects. It would lead us too far to enumerate all the different modifications of this apparatus that have been recommended from time to time; such an undertaking would, moreover, be altogether useless, as the form of bandage must be adapted to each individual case.

3. THE SURGICAL TREATMENT OF DISEASES OF THE STOMACH.

Diseases of the stomach are treated by surgical methods either in order to bring about a radical cure of some disease of the organ that cannot be cured in any other way, or in order to remove certain dangerous or very troublesome symptoms. It is self-evident that operative interference for either purpose is permissible only in those cases where internal therapy is unable to accomplish anything.

In order to proceed surgically a precise diagnosis must be made. It is the task of the internist to make a most exact diagnosis, in order to know in good time what the precise indications for surgical interference are. It is the task of the surgeon, on the other hand, to improve his methods of operating and to determine which is the best surgical procedure in each individual case.

The diagnosis of diseases of the stomach has made great progress within the last ten years; at the same time it is not perfected to such a degree that we are always able to determine sufficiently early when a case of stomach disease should be operated on, nor are we able in each case to determine in advance what would be the most profitable and the most suitable surgical procedure. In many cases conditions are found when the abdomen is opened that were unrecognized before and that call for a complete change in the original plan of surgical procedure.

It will be impossible, of course, to enter into all the details of surgical interference in diseases of the stomach; all that we will describe are the general indications for surgical treatment in this class of diseases. We will also briefly discuss the most important operations. Among these are resection of the pylorus or pylorectomy, gastro-enterostomy, divulsion of the pylorus, and pyloroplasty.

Among the diseases that call for surgical treatment the different stenoses of the pylorus are unquestionably the most important ones, particularly those that prevent the stomach from propelling a sufficient amount of food into the intestine. Those cases in which the internal treatment, including physical methods, like massage, electricity, methodic lavage, etc., do not lead to the goal, should undergo operation as soon as possible.

These stenoses may be of different origin, and an operation must be performed to suit the primary cause of the trouble, and also a variety of other circumstances. Stenoses may be divided into two groups—benign and malignant.

One of the most frequent causes of benign stenosis is cicatricial contraction of the pylorus. Such contractions frequently follow round ulcer of the stomach, and occasionally poisoning by acids or other eroding substances. Quite frequently tuberculous or syphilitic ulcers lead to stenosis. Finally, there may be simple hypertrophy of the musculature of the pylorus, leading to a narrowing of its lumen; or again peritoneal adhesions in the region of the pylorus or in the first few inches of the duodenum may lead to stenosis.

Mintz[1] has reported a case in which simple circumscribed adhesions of the pylorus with neighboring organs, followed by immobility of the pylorus, led to a severe ectasy of the stomach, with all its serious consequences. In this case a dilatation of the stomach of very high degree was found. Internal treatment was pursued for four weeks, and the stomach washed out daily. No benefits accrued to the case from this therapy, and so a laparotomy was performed. After opening the stomach it was found that the pyloric orifice was open, but that the posterior wall of the pylorus was adherent to its surroundings. A horseshoe-shaped scar was found on the mucous membrane of the posterior wall of the pylorus. The stomach was enormously dilated. In this case there was only a relative occlusion of the pylorus caused by an adhesion and by immotility of the pylorus that resulted therefrom. The patient died from peritonitis a few days after the operation.

Wiesinger[2] also reports a case in which stenosis of the pylorus was caused by a cicatricial band. The patient was a man of sixty-four in whom symptoms of stenosis of the pylorus appeared. No clue as to the origin of this lesion was discovered. A peculiar feature of the case was that the symptoms of stenosis became exacerbated and then improved again, and that lavage of the stomach, which was performed for a long time, remained without effect. A malignant neoplasm was positively excluded, and the operation revealed that no tumor existed. The laparotomy, however, showed that a cicatricial band extended from the anterior surface of the liver around the pylorus, back to the liver, without at the same time being adherent to the pylorus itself.

Benign tumors of the region of the pylorus may also lead to severe degrees of stenosis with secondary ectasy of the stomach; such tumors are, however, rare. Malignant tumors, as carcinoma of the pylorus, are the second group that may lead to stenosis of the pylorus, followed by ectasy.

If the diagnosis of stenosis of the pylorus with secondary ectasy of the stomach has been positively made, it will be necessary to decide whether this stenosis is benign or malignant. It is of great importance for the subsequent surgical procedure and also for the diagnosis of the case to decide whether the stenosis is due to a cicatrix from ulceration, to perigastric adhesions, to a benign tumor, or a carcinoma. The character of the surgical inroad will depend on the conditions present. The result of the operation, moreover, is not the same, even if we succeed in every case in removing the cause of the stenosis, whether it be a cicatricial ulcer, a benign or a malignant growth, or possibly a constricting band. In benign stenosis we will frequently, though not, of course, always, bring about a complete cure by removing the stenosis; in malignant stenosis this has never occurred. Billroth[3] has introduced the operation of pylorectomy, and it is possible with this operation to remove the carcinoma completely, and in this way cure the stenosis and its sequelæ. This, however, does not constitute a complete cure of the disease, for, as a rule, cases of carcinoma are operated on at a time when ectasy has developed, when free hydrochloric acid is absent from the stomach-contents—in other words, when the mucous lining of the stomach is more or less atrophic. This secondary atrophy that is caused by cancer persists after the operation. Unfortunately, the gastric juice has not been examined in many cases in which resection of the pylorus was made for cancer. An examination of the gastric juice before and after resection would certainly furnish much interesting and important information. The analyses that have been made so far seem to show that in all cases in which this operation was performed the production of gastric juice was not only not restored, but, on the contrary, gradually altogether suppressed.

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[1] *Zeitschr. f. klin. Med.*, vol. xxv., p. 128.

[2] *Therapeut. Monatsh.*, July, 1895; *Aerztlicher Verein zu Hamburg*, session of April 26, 1895.

[3] *Wien. med. Wochenschr.*, 1881, Nos. 6, 51; *Wien. klin. Wochenschr.*, 1891, No. 34; *Verhandl. d. X. internationalen Cong.*, vol. iii.

So far only a few cases (Rosenheim,[1] Boas[2]) have been observed in which removal of a carcinomatous pylorus improved the secretory powers of the stomach so that free hydrochloric acid, which was absent before, reappeared after the operation. Unfortunately, the reports do not tell us whether the absence of free hydrochloric acid was constant before the operation, nor whether a sufficient number of analyses of the stomach-contents were made to determine this point with absolute certainty. The very fact that free hydrochloric acid appeared after the operation demonstrates that the stomach mucosa can possibly have been completely atrophic. Conditions as favorable as this will probably be found in exceptional cases only. In the majority of instances the operation will be performed at a time when the changes in the mucous lining of the stomach are beyond repair—when atrophy of the glands has occurred. Notwithstanding all this, patients seem to recover quite rapidly after a successful operation for resection of the pylorus. This need not surprise us, for after resection the dimensions of the stomach, as a rule, become normal, the motor power of the stomach improves until it becomes normal, excepting in cases where the muscularis was atrophic or degenerated. If the stomach regains its normal motility, this constitutes a great advantage to the patient, for now the small intestine can vicariously assume the peptic function of the stomach ; nevertheless we can hardly speak of a complete cure even if the carcinoma is successfully removed. The possibility of a cure in cancer of the pylorus exists only in those cases in which the operation is performed at a time when no deep-seated changes of the mucous lining of the stomach have occurred. Unfortunately, carcinoma of the stomach is rarely operated on at so early a stage.

Aside from other difficulties, as wide-spread adhesions, great extent of the carcinomatous growth, secondary nodular metastasis in other organs, and other circumstances, a complete restitution to normal can hardly be expected after resection of the carcinomatous portion of the organ. This applies even to the most favorable cases.

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[1] *Berlin. klin. Wochenschr.*, 1895, No. 1 ; see *Verhandl. d. Vereins f. innere Med.*, and *Deutsch. med. Wochenschr.*, 1895, Nos. 1–3.

[2] *Ibid.*, 1895, No. 5, supplement No. 18.

In any case of stenosis of the pylorus that has led to secondary dilatation of the stomach in which internal therapy is unable to relieve or cure, the obstacle must be removed by surgical means.

Stenosis of the pylorus due to adhesions is most amenable to surgical treatment. Occasionally all that is needed is to separate the constricting membranes. Sometimes adhesions of this character cause severe disturbances. It is not necessary that the symptoms of ectasy appear in every case. Hahn[1] has recently reported a case in which an operative removal of these adhesions was followed by a complete cure. His patient was a woman who since her fifteenth year had been a sufferer from frequent hemorrhages of the stomach. All symptoms pointed to adhesions in the region of the stomach. The patient's distress was so great that she begged for an operation. Hahn performed a laparotomy and found nothing abnormal between the stomach and colon excepting five rather large adhesive bands. He placed double ligatures around these five adhesions, separated them, and from that time on all the symptoms disappeared.

Hernia of the linea alba, even preperitoneal lipomata, may cause serious gastric disturbances, and most urgently call for surgical interference.

Stenosis of the pylorus caused by cicatricial narrowing or by benign tumors may be completely cured by resection of the anterior portion of the pylorus—so-called pylorectomy. Quite a number of cases are on record in which resection of the pylorus was performed for cicatricial stenosis. Mintz,[2] in a paper that he has recently published, has gathered all the cases of resection of the pylorus for cicatricial stricture that have been published so far ; 31 cases are on record, 17 of which were cured—that is, 54.8 per cent. In the cases that died there was collapse or peritonitis. Statistics of this kind, of course, do not allow us to formulate any definite judgment in regard to the value of resection in cicatricial stenosis of the pylorus. The experience and the skill of each individual operator will naturally decide the issue ; the time at which the operation is performed, the general strength of the patient, and other circumstances will all have to be considered. In many cases the operation was performed too late—that is, at a time when the strength and the vitality of the patient were too far reduced to allow him to sustain the shock of an operation. Pylorectomy is probably preferable to gastro-enterostomy in cases of benign stenosis of the pylorus, because this operation removes the obstruction at once. It is a radical operation, and leads to a complete restitution to normal, whereas gastro-enterostomy simply forms a new channel for the gastric contents that leads around the obstruction. At the same time a number of valid objections may be advanced against resection of the pylorus in benign stenosis. In many instances the strength of the patient will hardly permit so long and so difficult an operation as resection; in other cases resection of the pylorus is impossible because the pylorus is adherent to its surroundings. In many instances again there is great difficulty in connecting the large lumen of the stomach with the smaller lumen of the duodenum, so that ultimately a gastro-enterostomy has to be performed. In one case that Krönlein[1] reports the opening of the duodenum was so narrow after resection of the pylorus that a narrow sound could barely be introduced into the bowel.

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[1] *Deutsch. med. Wochenschr.*, 1894, No. 43.

[2] *Zeitschr. f. klin. Med.*, vol. xxv.

For all these and many other reasons the operation of pylorectomy for cicatricial stricture has become more and more unpopular—in fact, many surgeons, as Mintz, go so far as to declare that pylorectomy is never indicated or justified in these cases. In mild degrees of stenosis they prefer pyloroplasty; in severe cases of stricture, or if many adhesions exist, they perform gastro-enterostomy. I can hardly agree with those surgeons who condemn resection of the pylorus altogether in cicatricial stricture; if the general strength of the patient is good; if no adhesions exist; if the amount of cicatricial tissue and the extent of the cicatricial contraction are slight; and if no other contraindications against resection exist, I believe that resection, being the more radical operation and the one that restores normal conditions, is by far to be preferred. In many cases a laparotomy will have to be performed before the exact operation on the pylorus can be decided upon.

A second operation that can be considered besides resection is gastro-enterostomy. Wölfler[2] introduced gastro-enterostomy into surgery of the stomach. The object of this operation is to construct a connection between the stomach and a loop of intestine. A new communication is in this way formed between the stomach and the duodenum or the jejunum, so that the stomach-contents can enter the intestine by a channel that goes around the obstruction. In this way the ingesta are more rapidly evacuated from the stomach. Wölfler first performed this operation in cases of carcinoma of the stomach in which complete resection of the diseased pylorus was no longer possible. Later the operation was also performed in benign stenosis of the pylorus. In cases of this kind the operation accomplishes even more than an avoidance of the stenosis and a more rapid evacuation of the ingesta. In benign cases the operation may lead to a disappearance of all the symptoms; this, of course, cannot be the case in carcinoma. As the ingesta after gastro-enterostomy no longer have to pass through the stenosed area, this portion of the stomach is no longer irritated; this leads to a complete cure of ulcers, inflammatory swellings, and other conditions that remained indifferent to all treatment before gastro-enterostomy was performed. The stomach may also resume its normal dimensions after gastro-enterostomy has been performed in cases of cicatricial stenosis of the pylorus, and the motor functions of the organ may also become normal. Sometimes this occurs rapidly and the restitution is complete; in other cases several months elapse before motility is normal, and certain disturbances of motility may be perceived for several months after the operation. In carcinoma of the pylorus all this is different. Mintz[1] has shown in his statistics that, as a rule, the general mechanism of the stomach improves after gastro-enterostomy, but that normal functional powers are never recovered. His studies of the functional results of operations on the stomach show that a certain degree of atony always remains after carcinoma of the pylorus.

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[1] *Centralbl. f. Chirurgie*, 1888.

[2] Wölfler, *Die Resection des carcinomatös erkrankten Pylorus*, 1881; *Wien. med. Wochenschr.*, 1882, No. 14.

Still less favorable results are seen in regard to the secretory functions of the stomach after gastro-enterostomy for carcinoma of the pylorus. An improvement in this function has never been seen after gastro-enterostomy; on the contrary, the powers of secretion seem to become more and more reduced after gastro-enterostomy, and finally to be lost altogether. If the carcinomatous pylorus is resected, it appears that atrophy of the stomach-glands may be arrested; it seems, therefore, that in carcinoma of the pylorus resection is to be preferred to gastro-enterostomy.

In cicatricial stenosis of the pylorus gastro-enterostomy seems to produce better results; here the motor function may be completely restored and the secretory function, even if hypersecretion exists, may become normal. Rosenheim[2] reports a case in which gastro-enterostomy was performed for cicatricial stenosis of pylorus in a case in which an enormous flow of gastric juice existed at the same time. After the operation the motor function of the stomach became normal and the gastrorrhea disappeared.

As far as we are able to formulate a judgment from the cases that have been observed so far, gastro-enterostomy leads to better results in cicatricial stenosis of the pylorus than pylorectomy. According to Mintz's statistics, recovery ensued in 71 per cent. of the cases, whereas in resection of the pylorus recovery occurred in only 54.8 per cent. It is to be expected that with an improvement of the technic of this operation the results will become still better, particularly if the operation is performed sufficiently early. We can see to-day that the results of gastro-enterostomy are better in simple cicatricial stenosis of the pylorus, and that, above all, the danger of this operation is less than that of pylorectomy. As an evidence of the comparative safety of gastro-enterostomy, I may mention that Hahn[3] did not lose one of his last 15 cases of gastro-enterostomy.

If wide-spread adhesions exist in addition to stenosis of the pylorus, or if the cicatricial thickening is very considerable and wide-spread, and if the general strength of the patient is much reduced, gastro-enterostomy is certainly to be preferred to pylorectomy, and is, in fact, the only operation in many cases.

Different authors have described a number of modifications of Wölfler's gastro-enterostomy; this question, however, is one of purely surgical interest, and we will not discuss it in this place.

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[1] *Wien. klin. Wochenschr.*, 1895, Nos. 16, 18, 20.

[2] *Sitzungsberichte der medicinischen Gesellschaft zu Berlin*, October 24, 1894; *Deutsch. med. Wochenschr.*, 1894, No. 44, p. 125.

[3] *Ibid.*, 1894, No. 43.

A third operation that must be considered in cicatricial stenosis of the pylorus is pyloroplasty. This operation was first performed by Heinecke-Mikulicz.[1] It consists in splitting the stenosis longitudinally and then uniting the wound transversely. This method, of course, cannot be applied to all cases, and cannot be performed where extended adhesions exist between the stenosed pylorus and neighboring organs, nor can it be performed where the stenosis is very extended, where the scar is too solid and rigid, or where an open ulcer exists. The results of this operation are favorable: according to Mintz's[2] statistics, 77.4 per cent. of the cases operated on recovered.

This operation is preferable to gastro-enterostomy because it is more rapidly performed, is generally less severe, and causes no changes in the gastro-intestinal function, like gastro-enterostomy; in fact, it restores normal physiologic relations.

On the other hand, it has this disadvantage, that it frequently cannot be performed because the case is operated on too late. If the stenosis is very great and extends over a large portion of the pylorus; if, at the same time, there is much hypertrophy of the muscularis or there are solid adhesions with neighboring organs; or, finally, if an uncicatrized ulcer exists, it cannot be performed. It is particularly suitable to those cases of cicatricial stricture of the pylorus that follow the swallowing of irritating substances; it is also a useful procedure in cicatrices that are due to ulcer, and in which no adhesions have formed.

It is impossible in many cases to decide in advance whether pyloroplasty can be performed or whether gastro-enterostomy must be done. As a rule, this decision can be rendered only after the abdomen has been opened.

There is another danger in pyloroplasty—namely, the possibility of a recurrence. It appears, it is true, from all that has been written on the subject so far, that a recurrence of the stenosis is rare in pyloroplasty; I have observed one case in which this occurred and in which symptoms of stenosis reappeared a short time after pyloroplasty had been performed. My case was one of ectasy of high degree in which a diagnosis of cicatricial stenosis of the pylorus was tentatively made. Professor Poppert performed pyloroplasty and brought about a complete cure of the case. The patient recovered quickly, gained rapidly in weight, the ectasy of the stomach disappeared, and a short time after the operation the patient was free from all distress and able to attend to his work. At the expiration of a year, however, some of the old symptoms reappeared and repeated attacks of hematemesis occurred. I saw the case several months after this, and found the same symptom-complex as before; in fact, the ectasy was greater than it had been before. The patient was extremely emaciated, and again all symptoms pointed to stenotic ectasy. Unfortunately, the patient refused a second operation; it is impossible, therefore, to decide whether the last stenotic symptoms were due to a recurrence of the former stenosis or whether a new ulcer and a new stenosis had developed in the mean time.

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[1] Fronmüller, *Operation der Pylorusstenose*, Inaug. Diss., 1886, and *Verhandl. d. Cong. d. Gesellschaft f. Chirurgie*, 1887.

[2] *Loc. cit.*

A fourth operation that can be performed in stenosis of the pylorus is divulsion of the pylorus, as recommended by Loreta.[1] This operation is performed as follows: An opening is made into the stomach close to the pylorus; into this opening the fingers or a sound are introduced, and the stenosis stretched and dilated. This operation has been attempted chiefly in Italy and in America. In Germany, justly, it has very few adherents.

Loreta's method is by no means so simple and so safe as it appears on first sight. In several instances hemorrhage and rupture of the wall of the pylorus have been observed. Durante,[2] for instance, reports a case of cicatricial stricture of the pylorus and of a portion of the duodenum in which he introduced Loreta's divulsor, and in which he tore the walls of the pylorus and of the duodenum; the tear in the duodenum was 7 cm. long. Another disadvantage of this method is the tendency to recurrence. Loreta himself observed recurrence of the stenosis three times within two to three weeks after the operation.[3]

[In our opinion Loreta's operation should never be performed. It is dangerous and ineffectual. Recurrence of the stenosis is almost certain to happen, and pyloroplasty is decidedly to be chosen in a case that appears suitable for divulsion.—Ed.]

I need not enter into a special discussion of the old operation of gastrostomy, or the making of an artificial gastric fistula. Simple gastrostomy is used for removing foreign bodies that have been swallowed and that cannot leave the stomach by the natural passages. Gastrotomy, with the creation of a gastric fistula, is indicated when food cannot enter the stomach—for instance, in stenotic processes involving the esophagus and the cardia. We refer to the section on Diseases of the Esophagus for the discussion of these operations.

A few words in regard to the various diseases of the stomach that call for surgical treatment and the different operations that are indicated in the several diseases. Of all diseases of the stomach, carcinoma is the one that calls for operation in the first place, particularly if the carcinoma involves the pylorus.

For this condition only two operations need be considered—namely, pylorectomy and gastro-enterostomy. It will depend on the general strength of the patient and on many other factors which of the two is to be preferred in any individual case. In general, the more favorable cases are suitable for resection, the more complicated and less favorable ones for gastro-enterostomy.

A carcinoma of the cardia cannot be extirpated. In a lesion of this kind gastrotomy must be considered, and in cases where this operation is impossible, owing to the contraction of the stomach, jejunostomy, as proposed by Hahn and Maydl, may be performed. Carcinoma of the body of the stomach is rare; if such a lesion exists, it may be possible to remove it by extirpation.

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[1] *Centralbl. f. Chirurgie*, 1883, 1884.

[2] *Ibid.*, 1893.

[3] Cyston (*Lancet*, March 23, 1895) recommends the following procedure as a substitute for Loreta's method. He orders the patient to swallow solid balls that are expected to act as bougies. He uses rubber balls of different sizes, and advises that they be swallowed in the morning immediately after breakfast. The results obtained so far, as was to be expected, are either altogether negative or at least doubtful.

Unfortunately, many physicians even nowadays prefer to let well enough alone in cases of carcinoma of the stomach, instead of advising an operation. This is due in great part to the comparatively unsatisfactory results that have so far been chronicled in operations for cancer of the stomach. It is true that a review of the general statistics on this subject shows that the results in regard to a cure are by no means brilliant; at the same time, a number of cases are on record in which the patient lived for a long time after the operation and enjoyed good health. Kocher, for instance, reports the case of a woman who enjoyed good health for five and one-half years after resection of the cancer. A patient of Wölfler's lived for five years after the operation without any digestive disturbances; at the end of that time a glandular metastasis developed. Hahn's patient had not suffered a relapse three and a half years after resection, and was in very good health.

Quite a number of cases are on record in which the subjects remained free from all distress for more than a year after the operation, and in which no relapses occurred within this time. In the majority of cases, it is true, relapses occurred much earlier.

It will be necessary to study carefully the conditions existing in each individual case before deciding whether resection or gastro-enterostomy is to be preferred; wide-spread adhesions, metastases, great loss of strength are absolute contraindications against resection; in cases of this character gastro-enterostomy should be performed. At the same time this is no radical procedure, for it does not remove the cancer: it removes only the symptoms resulting from stenosis of the pylorus; it may, however, lead to quite favorable results. Even though the cancer persists, the general nutrition and the strength of the patient frequently improve after gastro-enterostomy, although the operation is originally intended to do no more than to cause a more rapid evacuation of the ingesta from the stomach into the intestine by circumventing the carcinomatous pylorus. Another advantage of gastro-enterostomy is that the ingesta no longer have to pass the carcinomatous portion of the stomach; this may explain why the rapidity of the growth seems to decrease after this operation (Rosenheim).

The chief objections against the two operations that I have described, particularly against resection, are that they are too dangerous, and that the results are often too uncertain, for the patients were cured in only one-half of the cases. To these objections we might answer that, provided the diagnosis were made early enough and the patients were operated on at a sufficiently early stage of the disease, the outlook would not be so bad. If we should succeed in perfecting the technic of these operations, the outlook would be still better.

Kraske says correctly that all those writers who have gathered statistics on the subject have failed to judge carefully each case, and have contented themselves simply with enumerating the different cases and the results obtained. It is a very important matter to individualize in each case, and to weigh carefully the different features that it suggests. Kraske has critically reviewed a small but interesting number of cases in this sense. He operated 14 times for carcinoma of the pylorus, and performed resection 4 times; gastro-enterostomy, 10 times; of these 14 cases, 6 died—that is, 43 per cent. He divides his operations into two periods: those patients that were operated in the first period all died; those in the second, all recovered. On account of the unfavorable results obtained in this first period, Kraske refused for a long time to operate on any case of this kind that was sent to him for operation. These statistics teach us that the operation itself is not so very dangerous, but that the poor results obtained are due to the fact that the operation is performed at the wrong time.

Mikulicz[1] has also recently published some statistics that are instructive in this respect. This writer reports 103 operations of the stomach; among these there were 23 deaths. Of his 35 cases that were operated on in the first ten years, Mikulicz had 13 deaths—that is, 37 per cent. Among 68 cases that were operated later there were only 10 deaths—that is, 15 per cent.

It is of paramount importance that the diagnosis should be made as early as possible—that is, at a time when the carcinoma has not involved very large areas, has not formed metastases, and has not led to a reduction in the general strength of the patient.

Three operations must be chiefly considered in benign ectasies, particularly those that are due to cicatricial stenosis—namely, pylorectomy, gastro-enterostomy, and pyloroplasty. In cases where pyloroplasty can be performed, it should unquestionably be preferred. This operation gives the best results, and is the least severe inroad of the three. Unfortunately, most cases present themselves for surgical treatment too late for this operation. Pylorectomy frequently fails because extensive adhesions exist or the cicatricial tissue involves too much of the pylorus; in other cases the loss of strength is so great that pylorectomy cannot be performed, and the quicker and less serious operation of gastro-enterostomy must be performed.

From all that we have said it will be seen that ectasy of high degree caused by narrowing of the pylorus calls for surgical treatment, provided internal treatment is futile or a carcinoma exists which, *a priori*, renders all treatment by medication fruitless. In cases of primary or atonic ectasy surgical interference is less positively indicated and justified. Bircher[2] was the first to recommend operative treatment in this form of ectasy; he has published 3 cases of this character that he operated on by a method of his own invention. The operation of Bircher consists in reducing the capacity of the stomach by constructing folds in the stomach-wall and sewing these reduplications together (gastroplication).

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[1] *XXIV. Cong. d. Deutschen Gesellschaft f. Chirurgie*, 1895; see *Deutsch. med. Wochenschr.*, supplement, 1895, No. 13.

[2] *Correspondenzbl. f. Schweizer Aerzte*, 1891.

This method has not been adopted. The only surgeons who performed this operation besides Bircher were Weir[1] and Brandt.[2] Bircher's method must be considered irrational and unphysiologic, for even in the most favorable cases it can simply bring about a reduction in the size of the stomach for a short time; this reduction will always be transitory, and the disease proper—the atony—will not be remedied in any sense. The best that can be expected of this operation is, therefore, transitory relief of some of the most urgent symptoms.

[Bircher's operation of gastroplication in atonic ectasy has been done repeatedly by Roswell Park, and sometimes with good results. Park believes that the operation is to be recommended in certain cases. —Ed.]

Baudouin[3] has proposed a much more rational procedure—namely, to perform gastro-enterostomy in all primary cases of dilatation of the stomach that are at all advanced. So far only very few cases are on record in which gastro-enterostomy has been performed for primary ectasy of the stomach. Jeannel[4] reports a case of this kind that terminated fatally. Kleef,[5] however, reports a second case in which gastro-enterostomy was performed with good results.

Rydygier[6] also postulates that any form of obstinate dilatation of the stomach that is well developed must be considered suitable for operative interference, particularly if the condition remains uninfluenced by ordinary therapeutic measures. In atonic ectasy no other method is probably so suitable and applicable as gastro-enterostomy.

There are a few other rare operations that may possibly be indicated in conditions of this kind. Some of them we have already mentioned above—namely, for instance, separation of adhesions existing between the stomach-wall and neighboring organs. I have already mentioned a case of this character that Hahn reports. Here very severe symptoms urgently called for an operation, and the separation of the adhesion led to an immediate relief of all the patient's complaints.

Hacker[7] also reports 2 cases in which he brought about a cure by the simple separation of the strands of adhesive tissue that he found. Landerer[8] reports 3 other cases in which the stomach was mechanically fixed. The patient suffered violent gastric distress, but as soon as the adhesions were severed, all the symptoms disappeared completely.

As we have repeatedly stated, stenosis of the pylorus consecutive to ulcerative cicatrices is a common condition, and most frequently calls for surgical interference. Occasionally an ulcer that has undergone cicatrization, but has not led to stenosis of the pylorus, may have to be treated surgically. The symptoms that indicate that so radical an interference as a surgical operation is necessary are violent and frequently recurrent gastric hemorrhages that endanger the life of the patient.

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[1] *Centralbl. f. Chirurgie*, 1892.

[2] *Ibid.*, 1894, No. 16.

[3] *Progrès médical*, 1892.

[4] *Archives provinc. de chirurgie*, 1893.

[5] *Berlin. klin. Wochenschr.*, 1893, No. 44.

[6] *Wien. klin. Wochenschr.*, 1894.

[7] *Ibid.*, 1887.

[8] *Münch. med. Wochenschr.*, 1894, No. 39.

If the disease is very obstinate and intractable, or if perforation occurs, surgical help must be summoned at once. So far only a few cases are on record in which conditions of this kind were treated surgically. If the ulcer can be found, the whole involved area may be excised. Czerny,[1] Cordua,[2] and Mikulicz[3] have reported cases of this kind. If the ulcerated area is adherent to the surrounding tissues, as is almost invariably the case in ulcers of the posterior wall, the diseased area cannot, of course, be excised, and the ulcer must be cauterized.

[Mayo Robson, Roswell Park, and others have recently operated on numerous cases of chronic round ulcer that had proved resistant to ordinary methods of treatment. The reasons for operation have not been merely threatening perforation nor extensive hemorrhage, although, of course, these elements are considered, but also, in cases in which the ulcer is obstinate or recurrent, with marked delay in the healing processes, they recommend the excision of the ulcer and the cicatricial tissue surrounding it.

From personal observations in the results of this work it is felt that surgical treatment is to be commended in a limited number of cases of the kind described; and although a longer time for observation is needed before reaching a positive conclusion regarding a recurrence, it may be said that so far no tendency toward recurrence has been observed. It must still be held that surgery is applicable to exceptional cases only, and for the majority of cases of peptic ulcer the methods of treatment before described are the best.—Ed.] Dunin[4] recommends gastro-enterostomy in all those cases in which there is hemorrhage and in which the bleeding spot cannot be found. He claims that ulcers of this character, particularly if they are situated near the pylorus, heal much more rapidly if an artificial passage is created from the stomach into the intestine and the food no longer passes over the ulcerated places. Hahn goes still further and recommends jejunostomy; by this operation the stomach is completely isolated.

If perforation of a gastric ulcer occurs, surgical interference is urgently indicated. Pariser[5] has recently formulated some statistics that include all the cases of this kind that have so far been reported; there are in all 43 cases and 11 cures. The ultimate result will depend on the time at which the operation is performed, and the sooner after perforation the abdomen is opened, the better the prognosis; if more than ten hours are allowed to elapse after perforation, a successful issue is doubtful. Pariser claims that one of the chief conditions for a favorable result is the accessibility of the perforated ulcer; if the perforation can be rapidly reached and the stomach is not too full and its contents not decomposed, the prognosis is fair.

Pariser further calls attention to the fact that we are frequently dealing with double ulcers; both may be perforated at the same time, and one of the perforations may be overlooked. The chief place of predilection for a perforation of this character seems to be the anterior wall of the stomach, chiefly the cardiac region. The surgeon should look for the perforation in this part of the stomach-wall first. The reason why perforation seems to occur more frequently in this area than in any other is that adhesions only rarely form in this part of the organ. Ulcers of the anterior surface of the stomach are much more rare than of the posterior wall. Gould[1] has collected a number of cases of ulcer of the stomach, and finds that only 2 per cent. of those that were found in the posterior wall became perforated, whereas of the ulcers that were found in the anterior wall, 85 per cent. became perforated. Only a relatively small number were found in the latter position. Ulcers that perforate through the posterior wall usually lead to the formation of a circumscribed abscess, whereas those that perforate through the anterior wall usually perforate directly into the peritoneal cavity. We see, therefore, that ulcers of the anterior wall are much more prone to perforate than ulcers of the posterior wall.

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[1] *Arch. f. klin. Chirurgie*, 1884.

[2] Debove and Rémond, *Traité des mal. de l'estomac*, 1883.

[3] *Deutsch. med. Wochenschr.*, 1892.

[4] Quoted from Mintz.

[5] *Deutsch. med. Wochenschr.*, 1895, Nos. 28, 29.

The only chance for spontaneous recovery is given if the stomach is completely empty at the time of perforation. Under these conditions circumscribed peritonitis with adhesions develops. Pariser observed a case of this character that recovered spontaneously without any surgical interference.

If perforation occurs and the physician is not quite certain whether or not the stomach was empty at the time of the accident, laparotomy should be performed within ten hours. I need hardly emphasize that the administration of any food *per os* is strictly contraindicated even if the stomach was completely empty when the accident happened. It may appear superfluous to call particular attention to this self-evident precaution, and I would refrain from doing so had it not been violated in a number of instances that are on record. I mention it particularly so that similar error may be avoided in the future.

The operation, of course, consists in looking for the perforation and in closing it; the peritoneum should then be thoroughly cleansed.

In conclusion, I will mention a group of cases that call for surgical interference—namely, cases in which erosion of the mucous lining of the stomach has occurred, and in which, consequently, the patient is unable to take any food by mouth. In cases of this kind Hahn advises jejunostomy, and this operation may be performed.

It will be seen from this brief review that surgical interference is quite frequently called for in the treatment of diseases of the stomach. It is true that for the present the precise indications for the different operations are not strictly defined, and that the results obtained are in many cases not so satisfactory as might be desired. We may look forward, however, to the day when the diagnosis of these different affections will be perfected, when the nature of the different diseases will, in consequence, be recognized at a much earlier stage of development, and when, finally, the technic of stomach surgery will be so much improved that the results will be more favorable and more satisfactory.

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[1] S. Jowers, "A Case of Perforated Gastric Ulcer treated by Laparotomy: Recovery—Remarks," *Lancet*, 1895.

[When it becomes necessary to resort to surgical intervention in any stomach-disease, it would seem to be for the best interest of patients that the matter of feeding and the general management of the case should be under the direction of some one especially qualified to deal with diseases of the stomach. No food of any description should be allowed for several days after the operation, and generally none for the lapse of a week's time. Even water and ice should be prohibited for several days, water being meantime introduced in the form of enemata of normal salt solution. There are a number of questions likely to arise, as, for instance, vomiting, meteorism, constipation, and the use of anodyne, in which the opinion of a competent internist is desirable. —Ed.]

SPECIAL DIAGNOSIS AND TREATMENT.

INTRODUCTION.

In the general part of this volume we have discussed the methods for examining the stomach when it is diseased. We have also summarized and described all those pathologic symptoms that occur in different diseases of the stomach, and have discussed their significance. In this second part we will discuss the different diseases of the stomach that we encounter in practice.

We have repeatedly called attention to the fact that physical methods of examination are not sufficient. Formerly these were exclusively employed, but nowadays we need more. In every case of dyspepsia, whatever its origin, the gastric secretion, the motility, and the absorptive powers of the stomach should be carefully investigated. A progressive physician should not be content with merely giving the disease a name.

I will illustrate my meaning by an example: If, for instance, we are able positively to diagnose a carcinoma from the history of the case, the course of the disease, and the results of palpation and other manipulations, this is not sufficient. In one case the carcinoma will be accompanied by a great deficiency in hydrochloric-acid production; in another the hydrochloric-acid production will be just sufficient to saturate all the affinities of the proteid introduced; in a third case, possibly, we may find an overproduction of hydrochloric acid. Similar variations may be seen in the motor powers of the stomach in different cases: in the one it may be normal, though the production of hydrochloric acid be very much reduced; in the other it may be greatly deficient.

I need hardly mention that cases that differ in this respect naturally offer a different prognosis. The treatment of these cases, moreover, will be different according to the state of the functional powers of the stomach. In every case, therefore, of serious stomach-disease, or, better, of dyspepsia, the physician should gain all the information he can in regard to the secretory, motor, and absorptive powers of the stomach, provided, of course, the introduction of the stomach sound is not contraindicated. This is the only way thoroughly to understand the disease and to gain a clear insight into the nature and the intensity of the functional disorders present in any given case.

I use the term dyspepsia, although I am aware of the fact that its meaning is rather indefinite. This term was at one time very popular in medical nomenclature, but later fell into complete disuse. I do not use it to signify deficient or reduced digestive powers, but to indicate that the digestion in general is disturbed and abnormal.

This disturbance of the digestion may become manifest in many different ways: it may take place either in the sphere of gastric secretion, or of motility, or of absorption; one of these functions alone may be perverted or all of them may be perverted at the same time. Again, the perversion may extend merely to the sensory functions of the stomach. Leube's original definition of nervous dyspepsia included only those cases in which the chemism of the stomach was normal; the motor powers were also fully sufficient, but the patient complained of a series of subjective disturbances at the time of digestion. This form of dyspepsia, therefore, is a sensory neurosis.

I need hardly show in detail why this definition of nervous dyspepsia is altogether too narrow to fit our present views. We know to-day that, aside from pure sensory neurosis of the stomach, a great many real disturbances of the secretion of gastric juice and the motility of the organ may be caused by lesions involving the nervous fibers of the stomach alone.

Dyspepsia really means a disturbance of digestion. This disturbance may be of different character and origin; it may be dependent on anatomic changes in the stomach, it may be purely functional, it may be nervous. The only way in which to decide whether we are dealing with a sensory disturbance alone or whether the dyspeptic symptoms of the patient are caused by disturbances in the secretion of gastric juice, the motor powers of the stomach, etc., is to aspirate the stomach-contents and to examine it and then to make the diagnosis from the results obtained in this manner.

Within the last ten years aspiration of the stomach-contents for diagnostic purposes has been universally employed. A large number of facts have been revealed in this way that were unknown before; these facts explain many symptoms that were altogether obscure before, and at the same time give us many indications that are useful and profitable in the treatment of cases of stomach-disease. I need only refer, for instance, to the discovery that hyperacidity is almost constantly present in ulcer of the stomach, and that in carcinoma of the stomach the secretion of gastric juice is almost uniformly reduced; that there are certain perversions of gastric secretion due to nervous disorders; that gaseous fermentation can occur, and many other things. The analysis of stomach-contents has also taught us that one function of the stomach may be perverted alone, and that others need not necessarily be perverted at the same time. Formerly it was believed, on theoretic grounds, that the perversion of one function naturally led to the perversion of other functions, and that one function of the stomach was never perverted alone. It is true that under normal conditions secretion, motility, and absorption are in a certain sense related and go hand in hand; consequently the belief was justified, and was, in fact, harbored for a long time, that the deficiency in the production of gastric juice *eo ipso* led to a decrease in the motor powers and the absorptive powers of the stomach, and that, on the other hand, if the motility of the stomach was primarily reduced, the secretion of gastric juice and the absorptive powers of the stomach were under all circumstances damaged; this, however, does not necessarily apply to all cases. We frequently find one function very much reduced and another one considerably increased; cases are not at all rare in which there is a considerable degree of subacidity and in which the motility remains normal throughout. It is the fact that motility may remain normal, allow digestion and absorption of food to proceed in a normal manner, even though gastric secretion be disturbed.

It is the manifest duty, therefore, of every progressive physician to determine carefully the perversions of each function of the stomach. This should be done in every case of grave dyspepsia.

As a result of the more careful study of the different disturbances of individual gastric functions that occur in different forms of dyspepsia, the old clinical disease-pictures have been amplified and enlarged; in addition a number of new disease-pictures have been constructed that are based on purely functional disturbances, and lie outside of the ordinary anatomic classification of diseases of the stomach.

The picture of so-called nervous diseases of the stomach, of nervous dyspepsias, has been very much enlarged, particularly since we have learned to recognize that not only sensory and motor disturbances may originate from nervous disorders, but even disturbances in the secretory function of the stomach. In this manner we have begun to speak not only of sensory neuroses of the stomach, but also of nervous hyperacidity; we have, further, learned to regard hypersecretion as a neurosis. All this is unquestionably justified. Another question, however, arises: Is this manner of subdivision sufficient? are we justified in separating all functional disorders of the stomach, all forms of dyspepsia, into those diseases that are based on some tangible anatomic lesions of the organ and those that are purely functional in character? is it correct to designate the latter class as neuroses? I believe that a division of this character goes altogether too far; however desirable it may be to have an anatomic basis for every disturbance of function, we cannot say that we possess such a basis for the present in a large number of functional disorders of the stomach; at the same time it does not appear to me that we are justified in designating the latter class of disturbances neuroses, because we have not so far discovered the lesions of the stomach that cause them.

We are hardly justified in calling a disease a nervous disorder because pathologic-anatomic changes are absent, or, better, because we cannot find them; more is needed: we should be able to demonstrate and to prove that these functional disorders are really caused by nervous hyperstimulation or inhibition.

Leube, some eighteen years ago (in his dissertation on "Diseases of the Stomach" in von Ziemssen's *Hand-book of Special Pathology and Therapy*), expressed himself as follows in regard to neuroses of the stomach: "All those diseases of the stomach must be included under neuroses that consist in functional disorders of the stomach for which no anatomic basis can be found; in which, in other words, nothing is found to prove that the nervous system alone is not involved." I believe that this definition goes too far; there are, for instance, a number of cases of simple acute atony that rapidly recover under suitable treatment; no anatomic lesions can be discovered, and we hardly expect to find any; but there is no reason why we should call this disease a nervous disorder. If a case of tabes suffers from gastric crises, and if each time an attack occurs, as in the well-known case of Sahli,[1] acute hypersecretion appears, whereas in the intermediate periods the functions of the stomach are all normal, we are certainly justified in speaking of a purely nervous hypersecretion. On the other hand, we are not justified in designating every case of hypersecretion as a secretory neurosis. The same applies to hyperacidity. If a person suffers from oppression or cramps in the region of the stomach after some psychic excitement, and if, on examination of the stomach-contents at this period, a state of hyperacidity is found that is ordinarily not present, this will certainly be called nervous hyperacidity; but if, on the other hand, a person suffers from oppression in the region of the stomach every time he eats certain articles of food, and if it can be demonstrated that this feeling of oppression is due to hyperacidity, a reasonable doubt exists whether or not this disturbance is to be called a nervous disorder. The question becomes still more uncertain if, for instance, we find a persistent state of hyperacidity in subjects who have indulged in all kinds of excesses in eating and drinking, or if we find such a condition in chlorosis. Frequently we see cases in which hyperacidity of the stomach-contents is the one objective finding that must explain a number of subjective disturbances. It appears to me that we go much too far when we call hyperacidity of this character a nervous disorder because it does not fit into the frame of ordinary stomach-diseases that are constructed on some well-known pathologic-anatomic basis. It is much more rational to assume that in conditions of this kind we simply have been unable to discover the anatomic basis. It seems more conservative and more correct simply to remember that hyperacidity of this kind may occur in a variety of conditions and under different circumstances, and may produce certain symptoms.

Hyperacidity and hypersecretion are primarily functional disorders that may be due to a variety of primary causes; they may either originate independently,—that is, without any demonstrable cause,—or they may originate on a nervous basis or follow some anatomic disease of the stomach. Clinically, we can group all these forms together and designate them as we do atony or ectasy of the stomach. We feel justified in formulating different symptom-complexes, like atony and ectasy, and in considering them typical disease-pictures; the same can be done in hyperacidity and hypersecretion, and we will discuss them together as a peculiar perversion of gastric function. The fact that they originate from different causes and that their significance may be different in certain conditions does not alter this. Diabetes, for instance, is no uniform disease, but only a clinical complex of symptoms ; notwithstanding this we discuss this disease clinically as a peculiar morbid entity, although the pathologic-anatomic findings are either altogether negative or show some primary lesion of the pancreas or some other condition.

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[1] *Correspondenzbl. f. schweiz. Aerzte*, 1885, vol. xii.

Hyperacidity and hypersecretion may occur alone or in combination with other affections of the stomach. Clinically, they form a definite symptom-complex that is, in part at least, more or less characteristic ; this applies more to hypersecretion than to hyperacidity. I am of the opinion that they should rank as a definite clinical symptom-complex exactly like ectasy, atony, or diabetes, and that they merit separate discussion. My reason for entering into this discussion is that there is a tendency among modern students of the pathology of the stomach to classify all diseases of the organ according to the few pathologic-anatomic lesions that they have recognized so far. I consider this an artificial and a forced procedure, particularly as all those diseases that cannot be included in the different categories that have been artificially created are called nervous.

The prime object of clinical observation is to study the different disease-pictures that present themselves. Wherever we find a strictly characterized group of symptoms or some peculiar morbid condition, we should formulate a new disease-picture, even if no anatomic basis for the symptoms observed can be discovered. However desirable it may be to find the anatomic basis of each clinical disease-picture, we are forced in many instances to attach particular importance to the clinical disease-pictures that are presented at the bedside. I realize that I am establishing a new precedent when I discuss hyperacidity and hypersecretion separately, and when I formulate a distinct clinical picture that includes these two conditions ; at the same time I feel justified in doing this because the two states are so frequently met with. If we simply included hyperacidity and hypersecretion under catarrh or gastritis, the ordinary definition of gastritis would have to be changed. Most authors include gastritis among the neuroses, but I do not agree with this classification. While it is true that certain forms of gastritis originate from nervous disorders, this is certainly not the case in all instances. The nervous disorder is, at best, the chief etiologic factor in the majority of cases, and it seems to me that we would be going much too far were we to call all cases of gastritis nervous on these grounds. I do not doubt that in time a pathologic-anatomic basis will be found in most of these forms ; as a matter of fact, certain lesions have already been made responsible for many cases.

Erb and Charcot simultaneously and independently learned to separate primary spastic spinal paralysis from a large number of other diseases ; they did this purely from clinical observation. Their only reason for separating this one disease from a large number of other similar diseases was the peculiarity of the symptom-complex that they observed. At that time no one had discovered the pathologic-anatomic basis of this disease in the lateral columns of the spinal cord. The clinical picture of primary spastic paralysis attained the dignity of an independent clinical entity as soon as the above-named investigators succeeded in sharply distinguishing the symptoms of this disease from those of other similar diseases, and in establishing the individual features of this condition. It appears to me that the same applies to hyperacidity and hypersecretion in the case of stomach-diseases.

These arguments may explain my reasons for considering hyperacidity and hypersecretion, atony and ectasy, as independent diseases, and for discussing these different symptom-complexes separately; it may also explain why I do not adhere strictly to the anatomic classification of the different diseases of the stomach and attach more importance to clinical classification. In the case of disease of the stomach, as in many other fields of medicine, clinical observation has preceded our knowledge of pathologic anatomy. We physicians need not deny the individuality or the special features of any clinical picture because we are ignorant of the anatomic basis on which they originate. There are many diseases that we consider distinct clinical entities but in which we are altogether ignorant of the pathologic substratum; I need only refer, for instance, to chorea, epilepsy, and similar diseases. We are justified, notwithstanding, from the clinical point of view, in considering all these conditions separate diseases. Our knowledge of disease is not yet sufficiently advanced to permit us to adhere rigorously to the anatomic system of classification; we may expect, however, that many subjects that are still obscure will soon be cleared up, particularly in the case of the stomach, as no field of medicine has attracted so much attention and interest as this one. It appears to me that in arranging disease-pictures clinical observation should certainly be accorded first rank.

HYPERACIDITY, HYPERACIDITAS HYDROCHLORICA, HYPERCHLORHYDRIA, SUPERACIDITY, SUPERSECRETION, GASTRIC FLUX, CONTINUOUS SECRETION OF GASTRIC JUICE, HYPERSECRETION, GASTROSUCCORRHEA, GASTROXYNSIS.

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Compare also the standard text-books and monographs on diseases of the stomach.

Introductory Remarks.—Formerly it was believed that perversions of the secretion of gastric juice occurred only in the sense of a decrease of this function; nowadays we have learned to recognize that quantitative perversions may also occur in the sense of an increase. I believe that I was one of the first to call attention to these forms and to show that they occur with relative frequency. This increase in the secretion of gastric juice may be manifested in two forms—either as hyperacidity or as hypersecretion. Superacidity, hyperacidity, or hyperchlorhydria are terms that designate that abnormally acid gastric juice is being secreted during digestion, or, better, gastric juice that contains an abnormal quantity of hydrochloric acid. Here, as under normal conditions, the secretion of gastric juice occurs only when the gastric glands are stimulated by food. The gastric juice secretion, however, contains more hydrochloric acid than it should.

Hypersecretion, supersecretion, continuous secretion of gastric juice (Riegel), flow of gastric juice (Reichmann), are terms that designate an anomaly of the secretion of gastric juice in which the gastric glands pour out their secretion not only when stimulated by food, but even when the stomach is empty. If the stomach-contents of these cases is examined when no ingesta are present, active gastric juice will be found. This abnormal secretion may be transitory or intermittent, or it may last for a long time and be chronic.

It is necessary, therefore, to distinguish between hyperchlorhydria and hypersecretion; at the same time both conditions may occur together. If hypersecretion is present, hyperchlorhydria will frequently be found at the height of digestion.

In the literature references that I have quoted I do not separate the articles on hyperacidity from those on hypersecretion. I refrain from doing this because many of the dissertations refer to both forms of gastric anomaly. Many authors, in fact, do not separate the two forms, but consider them more or less identical; I am willing to concede that both forms are more or less related, and that possibly one form may merge into the other, or, again, that the two forms may be considered different only in degree. If the latter view is taken, excessive flow of gastric juice would constitute a greater degree of irritation of the gastric glands than hyperacidity. At the same time I do not believe that the two perversions, particularly when they are pronounced, can be considered identical. The disease-picture of hypersecretion is different in many respects from that of simple hyperacidity; certain differences in regard to the digestive powers of the stomach for certain articles of diet can readily be distinguished in the two forms. From a practical point of view, therefore, I consider a separation of the two disease-pictures indicated. Also from the therapeutic point of view I consider it important to separate the two, for while the method of treatment is the same in some respects, it differs considerably in others.

Most authors consider hyperchlorhydria and hypersecretion as neuroses, or, to be more precise, as secretory neuroses of the stomach. The perversions of gastric secretion can, without doubt, be nervous in character, for we know positively that the secretion of gastric juice depends on certain nervous influences. The well-known case of Sahli,[1] for instance, is undoubtedly one of nervous origin. His patient was a sufferer from locomotor ataxia with gastric crises; when he vomited, he raised abundant quantities of gastric juice that contained much hydrochloric acid, even if the patient had eaten nothing whatever for twelve hours preceding the attack. This phenomenon is a typical example of central irritation of the nerve-fibers governing gastric secretion. I might add, however, that a careful comparative study of the relation between gastric crises and hypersecretion has failed to reveal any uniformity or regularity between the two. Von Noorden[2] in particular has shown—and I have also determined by personal observation—that the secretion of gastric juice may differ greatly during these gastric crises.

Rossbach[3] has described another disease-picture that he calls gastroxynsis, and that is undoubtedly a true secretory neurosis. The disease manifests itself as follows: A perfectly well person suddenly sustains a violent psychic shock; soon thereafter a feeling of pressure in the region of the stomach, sour belching, nausea and vomiting of very acid material, occur. If the stomach-contents is examined, it will be found to contain an abnormally large quantity of hydrochloric acid. No one will, of course, doubt that this is a true secretory neurosis: that it is a case of hyperacidity of nervous origin.

Certain nerve-tracts may transmit impulses that stimulate the secretion of gastric juice either directly or reflexly; this we know both from clinical observation and from physiologic experiments, so that this fact is established without doubt. Beynard and Loye,[4] for instance, performed a conclusive experiment in the case of a criminal who had suffered capital punishment. They stimulated the vagus forty-five minutes after death had occurred, and were able to cause a secretion of gastric juice in the stomach. I also refer to the experiments of Pawlow and Schumowa that I have described above.[5]

There are, undoubtedly, a large number of cases of hyperacidity and hypersecretion that must be regarded as true secretory neuroses. The question arises, Are we justified in assuming a nervous origin in all cases? As far as I can see, we are not. Hyperacidity and hypersecretion are primarily functional disorders like motor insufficiency of the stomach. Motor insufficiency or atony may originate from some nervous disorder; at the same time every case of motor insufficiency is not necessarily a motor neurosis. It seems to me that we would be going much too far were we to declare every functional disorder of the stomach a neurosis because we were unable to find any anatomic basis for it. Motor insufficiency of the stomach, provided it does not lead to ectasy, leaves no anatomic traces, and at the same time it can hardly be considered a purely nervous disorder.

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[1] *Correspondenzbl. f. schweiz. Aerzte*, 1885, vol. xv.

[2] *Charité Annalen*, 1890.

[3] *Deutsch. Arch. f. klin. Med.*, vol. xxxiv.

[4] *Progrès médicale*, 1885, No. 29.

[5] See page 265.

We should consider a perversion of gastric secretion to be of a nervous origin only if we can positively demonstrate that this disturbance of function is due to the morbid stimulation or inhibition of certain nerve-tracts that lead to the stomach. We are not justified, on the other hand, in calling a disease of this character nervous because we fail to find anatomic changes. I think it is much more correct to class hyperacidity and hypersecretion as functional disorders that may originate from a variety of causes. In actual practice we see these two states arise from a number of conditions; in the case of hyperacidity, for instance, we can frequently demonstrate that some nervous excitation preceded it; in other cases we can determine that certain mechanical or chemical disturbances affected the gastric mucosa; in still other cases we see this condition in combination with other diseases, as ulcer of the stomach, chlorosis, etc.; and, finally, hyperacidity may be seen together with other symptoms of gastritis.

The same applies to hypersecretion. There are, undoubtedly, certain forms of this disease that are purely nervous in character; this is particularly the case in the periodic forms. In the chronic forms, on the other hand, the genesis is frequently doubtful. A large number of authors call the chronic form a neurosis; others again believe that it is the result of gastrectasy. This is not the place to enter into a detailed discussion of this question, for which I must refer to the section on Hypersecretion. There can be no doubt, however, that both forms, hyperacidity and hypersecretion, are peculiar disturbances of function, and that these disturbances of function lead to a peculiar typical and characteristic complex of symptoms. Like motor insufficiency of the stomach, these perversions may either appear alone as independent disturbances of function, or they may appear together with other diseases or in the train of certain other affections of the stomach. It is not impossible that occasionally a perversion of function that is purely nervous in character secondarily leads to anatomic changes in the stomach. If a case of this character is examined at a late stage, it is almost impossible to decide which was the primary and which the secondary factor.

The significance of this perversion of gastric secretion may, therefore, be different. In all cases, however, a series of peculiar symptoms is produced that it is important for the physician to recognize and to understand. It is just as important thoroughly to appreciate the significance of secretory perversions as it is to understand similar perversions in the motor sphere and to remember that both disturbances may originate from a variety of causes.

1. HYPERACIDITY, SUPERACIDITY, HYPERCHLORHYDRIA, HYPERACIDITAS HYDROCHLORICA.

We use the term hyperaciditas hydrochlorica to designate an increased secretion of gastric juice, or, better, of hydrochloric acid, during digestion. We know that the gastric mucosa excretes appreciable quantities of gastric juice only when it is stimulated. The normal stimulant for this function is the food ingested; as soon as food enters the stomach an abundant secretion of gastric juice, pepsin, and hydrochloric acid occurs; even in normal conditions more hydrochloric acid is secreted than is necessary completely to satisfy all the hydrochloric acid affinities of the albumin ingested. At a certain period of digestion, varying with the size of the meal, free hydrochloric acid will always be found in the stomach; this means that there has been an overproduction of hydrochloric acid; that more has been secreted than was necessary for satisfying all the affinities of the albumin. This plus we designate as free hydrochloric acid. Under normal conditions the quantity of this free hydrochloric acid fluctuates within certain narrow boundaries. Strictly speaking, we are dealing with hyperchlorhydria only in cases in which not only the total acidity is higher than normal, but in which the excess of free hydrochloric acid is also above normal. The total acidity of the stomach may be increased by other agencies—for instance, by the presence of organic acid; we should never, therefore, be content with alone determining the total acidity of the stomach, as we so frequently do. The free hydrochloric acid, too, should always be determined quantitatively.

Etiology.—To judge from my personal experience, hyperchlorhydria is a frequent functional disturbance. Bouveret, Jaworski, Mathieu, Remond, and others have expressed themselves in a similar sense. Other authors again claim that this symptom is a rare one.

I am unable to decide whether local influences play any considerable rôle in the causation of this condition. Fleischer seems inclined to this belief. If this author, however, draws the deduction from my reports on this condition that hyperchlorhydria is more frequently found in Hessia than in any other part of the country, I cannot agree with him. The patients in my clinic come from different regions—from Hessia, Nassau, Westphalia, the Rhine country, etc., and many of my private patients from very remote parts of the country, but I have not gained the impression from my observations that hyperchlorhydria is more frequent in certain provinces, as, for instance, Upper Hessia, than in others. It is possible that the difference in the frequency observed is in part due to the fact that we have performed diagnostic examinations of the stomach-contents in every case that presented even mild dyspeptic disturbances, provided, of course, that no direct contraindication existed for the passage of the stomach-sound. Other authors, on the other hand, limit their diagnostic examination of the stomach-contents to serious diseases of the stomach. In my private practice I have frequently seen patients that had been treated by many physicians for many years and in whom the stomach-contents had never been aspirated for diagnostic purposes, even though the whole symptom-complex made the diagnosis of hyperchlorhydria very probable. At the present moment, for instance, I have a patient in my private wards whose case has been diagnosed in many different ways; at the same time her stomach-contents had never been aspirated for diagnosis nor had an inflation of the stomach been performed, even though she was a sufferer at different times from violent dyspeptic symptoms. On careful examination I found that this patient was a sufferer from floating kidney on the right side, gastroptosis, and hyperchlorhydria, a combination of morbid conditions that is quite frequent in women.

It is impossible to formulate any definite statistics in regard to the frequency of hyperchlorhydria. Clinical studies of this condition have been reported in only very few instances, so that the material from which to draw conclusions is rather scanty. Until physicians learn to wash out the stomach-contents in every case that presents dyspeptic disturbances it will be impossible to determine how frequently hyperchlorhydria occurs, and what proportion of the secretory anomalies of the stomach it constitutes. Hyperchlorhydria is a perversion of function that physicians are more apt to see in private practice than in hospital practice; this is due to the fact that the symptoms of hyperchlorhydria rarely become so severe that the patients are forced to enter a hospital.

Age is certainly an important factor in the production of hyperchlorhydria. Young people are more frequently affected than old people; in the middle years of life the condition is also quite frequently encountered. No differences in its frequency in men and women can be determined; at least, that is my personal experience; if anything, the condition is more frequently encountered in women than in men, but this difference, if it exists at all, is insignificant. Jaworski has called attention to the frequent occurrence of hyperchlorhydria in Polish Jews; whether this is due to the peculiar diet of these people or to other conditions has not been determined.

To judge from my personal experience, chlorotic subjects are frequently afflicted with hyperchlorhydria. Oswald[1] some time ago published a report on an investigation of 21 cases of chlorosis in my clinic; only in one of these cases did he find values for hydrochloric acid that were near the upper boundary of the normal secretion. All others were considerably greater than normal; expressed in per cent., it was found that 85 per cent. of these cases of chlorosis were afflicted with hyperacidity and only 5 per cent. were normal.

Hyperchlorhydria occurs in all walks of life. In general, however, it appears more frequently in the better classes than among the laboring population. Nervous subjects seem to be particularly predisposed; persons who are excitable, who perform much mental labor, or women who are delicate and very nervous seem to be more predisposed to hyperchlorhydria than robust subjects. Neurasthenics frequently complain of dyspepsia that is due to hyperacidity. Von Noorden has repeatedly found hyperchlorhydria in cases of melancholia.

It is a well-known fact that psychic influences, violent emotions, sorrow, worry, mental overexertion, etc., may predispose to hyperchlorhydria, or even directly cause acute hyperchlorhydria; this is particularly the case in neuropathic subjects, in people who perform much mental labor, or who are very excitable. In cases of this kind any psychic emotion or other sudden nervous excitement may be followed

[1] *Münch. med. Wochenschr.*, 1894, Nos. 27, 28.

by acute symptoms that, on careful examination, will be found to be due to hyperaciditas hydrochlorica.

In other cases, again, the same factors may produce the symptom-complex that Rossbach has described. This author gives the disease-picture the dignity of an individual clinical entity, and calls it nervous "gastroxynsis." It appears to me, however, that gastroxynsis is no special disease, for the characteristic feature in this condition is periodic hyperchlorhydria. The fact that violent headaches are frequently complained of in cases that Rossbach would call gastroxynsis is hardly a sufficient reason for considering this combination of symptoms a special form of disease. Headaches are seen in many other severe attacks of hyperchlorhydria, as well as in other painful affections of the stomach. The symptoms of gastroxynsis, moreover, in all other respects resemble the symptoms of any severe hyperchlorhydria. That hyperchlorhydria exists in the cases Rossbach has described is stated by the author himself, and he even goes so far as to claim that the hyperchlorhydria is the primary cause of all the other symptoms.

It is possible that in many cases of so-called nervous gastroxynsis, particularly in those cases where the attacks set in when the stomach is empty (they usually occur some time after eating), were true cases of acute hypersecretion.

In other cases hyperchlorhydria may originate from some abnormal irritation of the mucous membrane that is kept up for a long period of time. It appears that in many subjects who are in the habit of eating too rapidly, of drinking large quantities of cold water, who indulge in alcoholic excesses, who use sharp condiments, and who do not masticate their food thoroughly, hyperchlorhydria is frequently seen. Some investigators claim to have observed that sudden changes in the ordinary and accustomed mode of life may produce hyperchlorhydria in certain people. It is impossible to determine whether gastritic processes are concerned in the production of this condition when any of the above-named abuses are indulged in; conditions of this character rarely cause the death of the patient, and consequently we are unable to control our diagnosis by postmortem examination and a determination of the anatomic conditions present. Slight inflammatory changes of the stomach, moreover, produce symptoms that are so little characteristic that we can rarely decide whether we are dealing with a purely functional disorder or whether a mild degree of gastritis exists.

It is generally recognized to-day that hyperchlorhydria is, as a rule, present in ulcer of the stomach. Some authors are undecided whether or not hyperchlorhydria is the cause or the effect of the ulcer; I am of the opinion (and refer for the details to the section on ulcer) that hyperchlorhydria is neither the cause nor the effect of ulcer. I think that the relationship between ulcer and hyperchlorhydria must be interpreted as follows: If hyperchlorhydria is present, much less favorable conditions for the repair of the injured mucous membrane of the stomach are given than if the acidity of the stomach is normal. If it is true, as many authors claim, that ulcer of the stomach is a very frequent condition in cooks, I think that the explanation for this occurrence must be sought in hyperchlorhydria; the frequent tasting of very hot food alone probably does not cause the ulcer, but it can produce hyperchlorhydria. If an irritated condition of this kind is once created, the ground is prepared for the development of an ulcer.

In many people hyperchlorhydria appears as soon as they eat certain articles of food or drink certain beverages; some people are afflicted with heartburn immediately after drinking strong coffee—they suffer from sour belching or may even have true attacks of cardialgia. On careful examination it will be found that these disturbances originate in a sudden and very transitory hyperchlorhydria.

Men who smoke too much also frequently suffer from hyperchlorhydria; some authors are inclined to include this form under acid gastritis. According to my personal experience, such attacks of hyperchlorhydria occur acutely after the excessive use of tobacco; even in men who are accustomed to smoke continuously, smoking a very strong cigar may occasionally cause acute dyspeptic disturbances. I had the opportunity of examining the stomach-contents of a number of cases in which this occurred, and found that hyperchlorhydria existed.

Hyperchlorhydria has also been reported in a number of cases of cholelithiasis; here it was found that the acidity of the stomach decreased as soon as the stone succeeded in passing. I have repeatedly made this observation. Some clinicians report similar gastric disturbances in nephrolithiasis.

This is essentially all that we know at present in regard to the factors that create a predisposition for hyperchlorhydria or cause this condition directly.

Symptoms.—The symptoms of hyperchlorhydria vary greatly. In a number of cases, which are not at all rare, hyperchlorhydria may exist and still no symptoms be complained of. In other cases the symptoms may be very mild or very severe: they may become so severe as to cause violent attacks of cardialgia. The symptoms always develop after eating; in fact, an intimate relationship exists between eating and the occurrence of an attack. The quantity and the quality of the food are very important. In some instances the symptoms begin very slowly, but later on increase in severity; in other cases, the symptoms appear suddenly and there may be veritable attacks that recur at short or long intervals. Quite a number of cases have been reported in which hyperchlorhydria occurred in acute attacks; the symptoms persist for a short time only; in other cases the perversion of function may persist for many years.

We can readily understand these differences. We must always remember that we are not dealing with an independent disease, but merely with a perversion of function that may occur under many different conditions. This perversion is seen only after the ingestion of food. Even in healthy subjects the secretion of gastric juice is dependent on the food, certain articles causing an excessive secretion, others a scanty secretion. Then there are individual differences in regard to the reaction of the gastric mucous membrane to different irritants. In some the secretion will be large, in others small. Finally, the intensity of the symptoms differs in certain individuals when the secretion of gastric juice is pathologically increased; in one subject a certain percentage of hydrochloric acid may cause disturbances that will not be noticed in another subject. We see, therefore, that the acidity of the stomach-contents is not proportionate to the intensity of the symptoms.

In some patients disagreeable symptoms appear after every meal, even after small meals; in others again they do not appear unless a very large meal is eaten, so that these patients suffer particularly after the large midday meal. In other cases, again, symptoms occur only after psychic excitement or the occurrence of some one or the other causes that we have enumerated above. When the stomach is empty, these patients are free from all distress, provided, of course, hypersecretion, spontaneous secretion of gastric juice, does not exist in addition to hyperchlorhydria. In cases of the latter kind the disagreeable symptoms may persist even after the normal time for digestion has elapsed. In pure hyperchlorhydria, however, the pain and the distress stop as soon as the ingesta are completely removed from the stomach.

In some cases symptoms appear after eating certain articles of food. It appears, however, that the occurrence of distress is not parallel to the digestibility of the food, so that an article that is easily digested may still cause more severe symptoms of hyperchlorhydria than one that is difficult to digest. The patients themselves are frequently astonished to find that they can eat very indigestible food without experiencing any distress, and, on the other hand, may suffer the most violent pain after eating some very digestible article of diet. The most plausible explanation for this phenomenon is that the irritability of the nerves of secretion varies at different times, so that at one time a light diet will cause an increased secretion of gastric juice, and at another time even a large and comparatively undigestible meal will not cause an abnormal irritation of the glands of the stomach. It is possible that in the latter case so much more free hydrochloric acid is combined by the food that subjective symptoms are absent.

We see from all this that the symptom-complex of hyperchlorhydria is variable. The intensity of the symptoms may fluctuate within wide boundaries, beginning in altogether insignificant and hardly perceptible symptoms and passing through all grades to the most violent distress. No single symptom nor group of symptoms can be considered pathognomonic for hyperchlorhydria; the disease-picture is, nevertheless, distinctly characterized in certain directions. Before discussing the different symptoms, therefore, I will briefly summarize the most important points in the symptom-complex of that form of hyperchlorhydria that is most frequently seen. There are certain cases that do not fit into the frame of this summary that can be separately discussed.

Patients that suffer from hyperchlorhydria, as a rule, do not create the impression of being very sick, and their nutrition is not reduced to any great extent. On the other hand, these subjects are rarely particularly well nourished or fat people; exceptions to this rule may, of course, occasionally be seen. The symptoms do not appear until some time after eating; the exact time at which they appear will depend on the amount of food that is eaten and the character of the diet; as a rule, the symptoms appear from one to three hours after a meal. In milder degrees the patients complain of thirst and of a feeling of fulness after eating; in other cases, again, there is real pain that may become so severe as to assume a spasmodic character. These symptoms persist for varying periods of time; in mild cases they disappear after half an hour; in other, more severe, cases they may persist for many hours. The patients frequently complain of acid belching and heartburn.

Vomiting is rare; only in those cases that are afflicted with violent cardialgic attacks may vomiting occur at the height of the attack. An examination of the vomit will reveal that it is very acid. The patients themselves complain that the taste of the vomit is excessively acid and burning. The quantity of the vomit and its general character will, of course, depend on the size and the nature of the meal eaten.

After vomiting has occurred, the patients, as a rule, feel relieved and the symptoms disappear. They seem to recover very quickly and to feel perfectly well after vomiting. In some people the symptoms that we have described occur only after the ingestion of certain articles of food; on the days on which they do not eat these particular things they have no symptoms whatever; in other cases the symptoms occur only after severe mental effort, after excitement, fright, or an attack of anger. In other cases again they appear more regularly, but in this class the symptoms are usually slight. Patients of this character complain of a feeling of pressure in the gastric region and burning or belching after eating. All these symptoms are transitory and disappear rapidly; they can frequently be made to disappear by eating something, and a very small quantity of food—for instance, an egg or a Zwieback—is often sufficient to relieve all distress.

During the night the patients are usually comfortable and suffer no pain. If symptoms similar to these that we have described occur repeatedly during the night, we are justified in suspecting that hypersecretion exists at the same time, or that the case is complicated in some other way.

The appetite is generally good. Many patients have a desire to eat something often, and, therefore, eat at frequent intervals, because they have learned from experience that the symptoms do not appear so readily if they do this. Occasionally canine hunger develops.

The external examination of the patient rarely reveals anything of importance. If the patients are examined in the intervals between the attacks of pain, it will be found that the region of the stomach is, as a rule, not sensitive to pressure. During the attacks of pain this is different; if the patients are examined during this time, it will be found that the region of the stomach is more or less sensitive to pressure. In other cases this sensitiveness to pressure may be so exaggerated that the slightest touch causes much distress. In contradistinction to ulcer of the stomach this painfulness is diffuse and extends over the whole gastric region. Occasionally it will be found that the region of the pylorus is particularly sensitive to pressure.

The above is a rough sketch of the general disease-picture of hyperchlorhydria. There are a number of deviations from this scheme in individual cases. It is, of course, impossible to make a diagnosis of hyperchlorhydria from the symptoms we have delineated; all that the occurrence of such a symptom-complex signifies is that we may suspect the presence of hyperchlorhydria. The only way in which to determine whether or not this suspicion is correct is to aspirate the stomach-contents and to submit it to a careful diagnostic analysis.

I will now proceed to the discussion of individual symptoms. The most important of the subjective symptoms are those of sensory irritation, and chief of these is the pain; in many cases, however, pain proper is completely absent. Many patients complain of slight discomfort or a feeling of pressure, a sensation of heat, and similar symptoms a short time after eating. They do not suffer from anything else. Other cases, again, for example, patients with chlorosis, complain of weakness, palpitation, loss of appetite. Gastric pain proper may be completely absent throughout the whole course of the disease.

In other cases again more violent symptoms are complained of; in fact, violent cardialgic attacks may occur. The latter do not recur regularly, but usually at long intervals. The patients seem to be perfectly comfortable and to suffer no distress on the intervening days.

If pain is present at all, it will always occur at the time of digestion, provided, of course, that hypersecretion does not exist at the same time. The pain never occurs immediately after eating, but always some time afterward, usually from one to several hours after the ingestion of food. The amount of food eaten and the character of the diet will determine the time at which the attacks occur; the more abundant the meal, the later, as a rule, the attack.

These paroxysms of pain are directly due to the abundant secretion of hydrochloric acid. In the beginning the hydrochloric acid that is produced by the glands of the stomach is immediately combined with the proteid. As soon as all the albumin affinities are saturated, free hydrochloric acid appears. The more abundant the secretion of hydrochloric acid, the greater, of course, the acidity, and the greater the likelihood of symptoms of this condition. The more albumin the meal contains, the more acid will be combined; consequently the pain or the attacks of cardialgia will appear at a correspondingly delayed period.

The interesting observation can occasionally be made in subjects that are afflicted with hyperchlorhydria that some of these patients suffer from severe gastric disturbances after eating a small meal, but if they eat a large meal, however, they frequently remain free from all distress. The explanation for this peculiar phenomenon is, of course, the following: the ingestion of a large amount of food combines a greater proportion of the hydrochloric acid secreted, and drinking large quantities of water dilutes the stomach-contents so that the accumulation of free hydrochloric acid is reduced. Inversely, we frequently see that cases of this character, at least when their symptoms first appear, can relieve the distress they feel by eating something more—a little albumin, a small quantity of milk. The *rationale* of this treatment is that the free acid is immediately combined. If they allow the pain to develop until it reaches a certain height, these measures will no longer relieve, for if the pain has reached a certain degree of severity, the patients are no longer able to eat anything. Even alkalis, which frequently relieve the pain very quickly, fail to act when the attacks have become intense. Notwithstanding the administration of these remedies the spasm continues; in fact, bicarbonate of soda, which is so popular, and justifiably so, frequently exacerbates the spasmodic pain. This is due to the fact that carbonic-acid gas is liberated and distends the stomach-walls. It frequently occurs that the administration of these remedies causes vomiting; this usually stops the pain and relieves the spasmodic movements of the stomach.

To judge from my personal experience it is, in general, rare to find cases in which such violent attacks of pain continue for a long time after each meal. As a rule, the intensity of the pain varies: on some days it will be slight, on others more severe. Occasionally attacks of pain recur at irregular intervals—even weeks may elapse between two attacks. Not only the amount of food eaten, but the quality of the diet, exercises an important influence on the occurrence of these attacks of pain. As a rule, we may say that pain appears more readily and more frequently after the ingestion of abundant quantities of amylaceous food than after an abundant meat-diet. In some subjects the paroxysms of pain occur more frequently after the midday dinner; in others, after the evening meal. We must assume that certain idiosyncrasies play a rôle. In some cases we see that attacks of pain occur as soon as a cup of coffee is taken, whereas tea, milk, and cocoa can be borne without causing any distress. In other cases I have frequently found that symptoms appear only if patients smoke immediately after breakfast or after dinner.

The symptoms of hyperchlorhydria that we have enumerated rarely appear suddenly, but usually develop slowly and gradually. At first the patients complain of a disagreeable sensation in the stomach; some designate the sensation as a feeling of pressure or fulness; others as a sensation of heat, of tingling, or of unrest in the stomach. In the milder cases these symptoms remain slight and the disagreeable sensations disappear after a short time. In other cases again the administration of soda or of a small quantity of chocolate, Zwieback, cakes, or something of that sort, will frequently stop the pain. In other cases again the pain grows worse and all the symptoms become exacerbated; there will be acid belching, heartburn, and, finally, spasmodic attacks of violent pain; the patients become utterly helpless, and the pain may grow so severe that the patients moan and groan. Many persons claim that in these violent attacks they can distinctly feel the stomach performing spasmodic movements. The explanation of this phenomenon is that an excessive secretion of hydrochloric acid will primarily cause spasmodic contractions of the pylorus, and that this will secondarily be followed by an increased peristaltic action of the stomach intended to overcome the obstacle that the contracted pylorus opposes to the passage of the stomach-contents from the stomach into the intestine.

These violent attacks are frequently accompanied by severe belching and heartburn. Patients complain of severe burning along the back; in others, the burning is felt in the esophagus. When belching is severe, a part of the hyperacid stomach-contents is propelled into the esophagus; this causes irritation of the mucous membrane of this organ, and indirectly painful sensations along the spinal column or in the esophagus itself. In very severe attacks violent headache is frequently complained of.

The duration of an attack of this kind may vary greatly. Mild paroxysms usually pass off very quickly, and last only from a quarter to half an hour. The more violent attacks may persist for many hours.

I have found that the appetite varies. There are certain cases of hyperchlorhydria in which the appetite is very good; in others—and these constitute the majority of cases—there is a normal and frequent desire for food, but the patients are unable to eat large quantities of food at one sitting. As soon as a small quantity of food is taken they feel satiated; at the same time they experience a desire for frequent meals. Occasionally there are sudden attacks of canine hunger combined with a feeling of weakness; as soon as a few morsels of food are swallowed these sensations disappear.

Many clinicians claim that thirst is also frequently exaggerated in these cases. To judge from my personal experience, I cannot corroborate this statement; at least not in the majority of uncomplicated cases. Those cases that are complicated by motor insufficiency or ectasy of high degree do, in fact, suffer from thirst even if hypersecretion exists in addition to hyperchlorhydria. In pure uncomplicated cases of hyperchlorhydria, however, I do not think that excessive thirst is the rule.

If, in an exceptional case, a patient of this character complains of excessive thirst, it will be only at the time of active hyperchlorhydria.

The action of the bowels varies and is frequently irregular. Many patients show a tendency to constipation; occasionally diarrhea and constipation alternate.

We will not discuss the objective symptoms of this condition. The general nutrition and the appearance of the patients with hyperchlorhydria are, as a rule, relatively good. Cases of this character are never greatly emaciated, nor do they ever present a cachectic appearance unless hyperchlorhydria is complicated with some other disease.

External examination of the stomach in pure uncomplicated cases of hyperchlorhydria rarely reveals any abnormalities, particularly if the examination is performed at the time between the attacks; there will be neither particular sensitiveness to pressure nor an abnormal distention of the stomach, nor succussion sounds, nor anything else.

If the stomach is examined at the time of the attack, or in cases that are complicated with some other disease, certain abnormalities will be discovered.

At the time of the paroxysms the region of the stomach will usually be distended. As a rule, the gastric region is sensitive even to slight pressure, or it may even be painful. The pain extends over the whole region of the stomach, but is felt particularly in the region of the pylorus, for here the most violent spastic contractions occur. Pressure on the region of the pylorus, therefore, usually causes pain. Occasionally the region of the pylorus hurts even without pressure; such cases are not altogether rare.

The final decision must be rendered by examination of the stomach-contents after a test-breakfast or a test-meal. If the stomach is aspirated six to seven hours after a test-meal, the stomach will be found empty unless complications exist; sometimes the stomach will be found empty after four or even three hours. Occasionally only a very small quantity of finely distributed food will be found at this time.

The same will usually be observed after a test-breakfast; after an hour the stomach will either be found empty or only a few cubic centimeters of a thin, finely divided mixture of food and gastric juice will be aspirated.

This proves that the motility of the stomach may be intact or even increased, notwithstanding the existence of hyperchlorhydria. We may say, therefore, that, as a rule, an increased secretion of gastric juice is accompanied by normal or even increased motility of the stomach.

I am unable to agree with those authors who claim that hyperchlorhydria is usually accompanied by a reduction in the motor powers of the stomach. I might say, on the contrary, that a person with hyperacidity usually digests his food very rapidly; there are, of course, exceptions to this rule, which are seen particularly in cases that suffer from very violent cardialgic attacks occurring chiefly after the ingestion of indigestible food, abundant quantities of amylacea, etc. In these cases spasmodic occlusion of the pylorus causes a retention of the ingesta; but even here the stomach will be found empty if the patient is examined within three hours after a test-meal.

Certain complications may, of course, exist that prevent the passage of the ingesta from the stomach into the intestine within the normal time—for instance, hypersecretion, ectasy, or dislocation of the stomach. In pure uncomplicated hyperchlorhydria, however, digestion, as a rule, proceeds rapidly, and the ingesta are quickly expelled from the stomach.

If the stomach-contents is examined with the ordinary color-reagents, it will be found to give very acid reaction. The test with Congo-paper alone may frequently lead us to suspect the existence of hyperchlorhydria from the intensely dark-blue color that the paper assumes. The final decision can, of course, be rendered only from the quantitative determination of hydrochloric acid. It is not sufficient to limit the examination of the stomach-contents to a determination of the total acidity, but the quantity of free hydrochloric acid should also be determined.

Both these values will be found increased. The average normal value for the total acidity is from 40 to 60 for a test-breakfast, and about 75 for a test-meal. In hyperchlorhydria the acid value after a test-meal frequently exceeds 100, and may attain 150, 160, or even more.

After a test-breakfast, when we might expect to find low values, the acidity may reach 100 and more. The quantity of free hydrochloric acid varies; it never runs parallel to the increase in the total acidity. We quite frequently find the total acidity increased, and at the same time only a small quantity of free hydrochloric acid. The most important point, of course, is the amount of free hydrochloric acid present. We can speak of hyperaciditas hydrochlorica only if the values for free hydrochloric acid are increased. Values of from 60 to 70 and 90 and above after a test-meal, and of 50 to 60 and above after a test-breakfast, are quite frequently found.

Organic acids have nothing to do with pure hyperchlorhydria. The same applies to gaseous fermentation; if the latter is present, we may suspect motor insufficiency.

The digestive power of the stomach is almost without exception very good; this can readily be determined by inspecting the stomach-contents and by performing an artificial digestion test with some of the gastric juice that is aspirated. As a rule, a small disc of albumin will be digested in a very short time. Absence or deficiency of pepsin is rare in cases of hyperchlorhydria.

If the patient eats only very small quantities of amylaceous food but an abundant quantity of meat, the amylacea are usually digested within the normal time, so that they disappear from the stomach at the same time as the meat. The increased amount of hydrochloric acid that is present in the stomach-contents necessarily accelerates the peptonization of albumin. The amylolytic power of the stomach is, however, not influenced in the same way by excessive hydrochloric acid; free hydrochloric acid, in fact, as we know, impedes the action of ptyalin and consequently hinders amylolysis. The sooner free hydrochloric acid appears in the stomach, the more will the amylolytic stage of digestion be abbreviated. If moderate quantities of amylacea are administered, the ptyalin in the stomach has plenty of time completely to convert all the starch, even though hyperchlorhydria exists, for a certain time must necessarily elapse before all the affinities of albumin are saturated and free hydrochloric acid appears. If, on the other hand, very large quantities of amylaceous food are ingested, the amylolytic stage of digestion in hyperchlorhydria will be found too short to produce a complete conversion of all the starchy food eaten; consequently a portion remains undigested, because the action of the ptyalin is stopped too early by the appearance of free hydrochloric acid; if this occurs, the ingesta may remain in the stomach an abnormally long time. Stagnation of stomach-contents is produced in this way, and if this condition becomes permanent, chronic hypersecretion of gastric juice may develop.

In those cases in which there is a continuous secretion of gastric juice the digestion of amylacea is still more impeded, for here a certain amount of hydrochloric acid is always present in the stomach ; to this amount is further added the quantity secreted as soon as food enters the stomach. Similar conditions obtain where motor insufficiency exists together with hyperchlorhydria. If motor insufficiency is present, the stomach will always contain old particles of food, and usually, in addition, free hydrochloric acid. As soon as more food is eaten, the secretion of hydrochloric acid is again stimulated, so that a still greater excess of hydrochloric acid will be present; consequently the digestion of amylaceous food is hindered to a still greater degree.

If the stomach-contents is aspirated in the latter class of cases immediately after a test-meal, the results of the analysis of this gastric contents will vary according to the contents of the stomach before the test-meal was administered. If the stomach was washed out before the administration of the test-meal, we will find different conditions than if this treatment was not instituted. At all events, the presence of large quantities of undigested amylacea in the stomach-contents and the evidence at the same time of good meat digestion will always lead us to suspect motor insufficiency, particularly if gaseous fermentation is present at the same time.

Absorption in the stomach is not disturbed in pure hyperchlorhydria. If the well-known iodid of potassium test is instituted, it will be found that, if anything, absorption is more rapid than in normal cases.

The urine presents no characteristic features. The greater the production of gastric juice, the greater the reduction in the acidity of the urine after meals. If there is abundant vomiting of gastric juice containing much hydrochloric acid, the acidity of the urine will also be reduced for many hours. Sticker's[1] experiments have shown that the abundant secretion of gastric juice may lead to a decrease of the chlorids in the urine. An excessive excretion of chlorids is only transitory, and can occur only if conditions are present that favor their retention in the stomach or their excretion from the organism (?). At the same time we are not justified in formulating any conclusions that can be utilized in differential diagnosis from the *absolute* amount of chlorin excreted in the urine.

The urine is occasionally cloudy and may have an alkaline reaction, particularly after violent vomiting. If this is the case, the phosphates are apt to be precipitated.

The subsequent course of hyperchlorhydria varies in different cases, and the degree of acidity itself may fluctuate within wide boundaries. The patients frequently feel perfectly well for days or weeks or even months, then again more or less severe symptoms may appear. It is true that if the stomach-contents is analyzed in the intervals of comparative well-being, mild degrees of hyperchlorhydria may be found. This hyperchlorhydria increases and will be found high at the time of the exacerbation of symptoms. Attacks of this character are frequently started by psychic excitement, by mental overexertion, by an irrational mode of life, and other factors. In some instances the primary cause cannot be discovered.

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[1] *Berlin. klin. Wochenschr.*, 1887.

In other cases, again, exacerbations of this kind occur after repeated excesses. The symptoms in this instance are either referred to the stomach alone or are of a more general character; the patients, it is true, complain of a feeling of pressure and of discomfort after eating, but this soon passes off; they are depressed, are not disposed to attend to their duties, they suffer from headache, insomnia, and similar symptoms. Cases of this kind frequently create the impression that they are sufferers from neurasthenia, and this diagnosis is often made; in fact, it is frequently difficult to determine which is primary and which, secondary. Occasionally an attack of migraine may occur, and hyperchlorhydria be present at the same time; the one, it appears, may be dependent on the other. Migraine, as a rule, is the result of hyperchlorhydria, and treatment instituted against the latter condition usually cures the former. There are still other cases in which hyperchlorhydria exists for a long time and causes no symptoms whatever; in the course of time, however, attacks of pain occur at irregular intervals. If these cases are carefully studied, it will be found that there is no great increase in the hydrochloric acid secretion at the time of the attack, but the food remains in the stomach an abnormally long time. Attacks of this kind are frequently distinguished by a peculiar feature—namely, by the fact that the attack of pain occurs later than usual: that is, not two to three hours after a meal, but many hours afterward. A patient of this kind may eat his supper at eight o'clock in the evening and wake up at midnight with pain that gradually increases. If vomiting occurs, the material raised consists of a very acid mass of undigested food that makes the teeth feel dull. We can assume that in cases of this kind there was hyperchlorhydria, and that, in addition, the acid stomach-contents was retained for an abnormally long time, so that ultimately a spasmodic attack occurred.

If the stomach-contents of such patients are examined for several days during the interval between the attacks and when they are not suffering any pain, it will be found that after a test-meal there is hyperchlorhydria, but that the amount of material found in the stomach after two or three hours is very small, and that after three and a half or four hours, or even before that time, the stomach will be found completely empty. It is probable that if this abnormally acid stomach-contents is allowed to remain in the stomach for a long time, hypersecretion may gradually develop. The retention of food in the stomach causes irritation of the mucous membrane and ultimately leads to a permanent secretion of gastric juice that does not stop until all food is removed from the stomach. Atony and ectasy of the stomach may also develop in this manner. In the most severe cases of this kind the stomach is probably never empty. Even here we cannot properly speak of hypersecretion, for this condition is present only if the stomach secretes gastric juice, even without any stimulation by ingesta. On the other hand, I do not doubt that cases of atony of this character, if combined with hyperchlorhydria, may ultimately produce a condition of the stomach in which the mucous lining secretes gastric juice, even without being irritated and stimulated by ingesta. [Strauss and Schuler think that in hyperacidity the contents of the stomach is found to be unusually large, mostly fluid, the specific gravity low, and amidulin present in unusually large quantities.—Ed.]

I will not discuss the relation between ulcer and hyperchlorhydria in this place, and must refer for this discussion to the section on Ulcer. I will only mention that patients with hyperchlorhydria seem to be particularly predisposed to ulceration of the mucous membrane of the stomach. It can hardly be considered a matter of chance that ulcer and hyperchlorhydria are almost without exception found together.

Prognosis.—The prognosis of uncomplicated hyperchlorhydria is favorable. The disease may be cured, and frequently is cured; even if the condition cannot be completely cured, it can at least be controlled by sensible dietary regulations and by sparing the stomach. If the patient presents himself when the disease has existed only for a short time, the prospect of a complete restitution to normal is much better than if the disease has existed for a long time; recurrences and relapses are, however, quite frequent. The symptoms may all disappear under appropriate treatment, and the patients be perfectly comfortable for several years; suddenly, however, the old symptoms may recur. If this happens, they are usually more severe than they were during the first period of sickness.

If hyperchlorhydria is complicated by atony or ectasy, the prognosis is less favorable. Cases of this kind, at all events, are very obstinate and seem to resist treatment. The possibility of a transformation of a simple hyperchlorhydria into a condition of the stomach in which there is a continuous secretion of gastric juice must always be kept in mind. If hyperchlorhydria is complicated with some other disease like ulcer, cholelithiasis, etc., the prognosis of hyperchlorhydria will correspond to the prognosis of these diseases.

The purely nervous forms are frequently very obstinate. The prognosis will depend on the primary cause; if it is possible to remove this, the disease may be cured.

Pathologic Anatomy.—We can hardly expect to find any pathologic-anatomic changes in genuine hyperchlorhydria, for this disease constitutes a simple perversion of function. As hyperchlorhydria *per se* rarely causes the death of a patient, we need not be surprised that no examinations of the stomach have been made in cases of this kind. Oestreich,[1] however, has reported a very remarkable case of hyperchlorhydria. His patient was a man of fifty-seven years who had suffered for years from intermittent hyperchlorhydria. Fourteen years before his death he had an attack of pneumonia, followed by thrombosis of one of the crural veins. Dating from that time he frequently had attacks of thrombosis that forced him to remain in bed; this depressed the patient very much, although he was otherwise an active man. Every time he was forced to remain in bed for some time peculiar attacks would occur consisting of violent paroxysms of pain in the region of the stomach and continuous vomiting. An analysis of the vomit revealed that it contained a very large percentage of hydrochloric acid. The patient finally died from an intercurrent pneumonia. An anatomic examination of the stomach revealed that everything was perfectly normal with the exception of a few slight erosions of the mucous membrane. Microscopic examination failed to reveal anything abnormal.

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[1] *Verein f. innere Med.*, meeting of July 1, 1895; *Wien. klin. Rundschau*, 1895, No. 31.

In cases, of course, in which hyperchlorhydria exists for a long time and without interruption, or where it is a symptom of gastritis, ulcer, gastrectasy, or other conditions, certain anatomic changes will be found. The first condition mentioned—namely, persistent uncomplicated hyperchlorhydria—has never been examined postmortem because, as we have stated, the disease rarely causes death. In regard to the anatomic findings in the other diseases named, we refer to the sections on these different stomach-lesions.

[It has been suspected, because of the accompanying anorexia, that jaundice decreases the gastric secretions, but this view is not supported by recent investigations.

Simitzky[1] found in experiments on dogs that when the bile-ducts were tied, the gastric mucous membrane secreted free HCl in excess. The superacidity disappeared in every instance with the disappearance of the jaundice. In one instance in which there was relapse of the jaundice the superacidity returned. It would appear that in animals bile retention increases the sensitiveness of the gastric mucosa to normal stimuli.—Ed.]

Diagnosis.—The diagnosis of hyperchlorhydria can be made positively only by a careful examination of the stomach-contents. The stomach-contents should be aspirated in all cases and a quantitative determination of the hydrochloric acid be formed. All the other symptoms, however pronounced, will only entitle us to suspect the existence of hyperchlorhydria, not to diagnose the condition with certainty. Of course, the repeated occurrence of pain in the region of the stomach, or the recurrence of attacks of pain at regular intervals a short time after eating, particularly if these attacks of pain are relieved by eating a little albumin or by taking some alkali, may lead us to suspect hyperchlorhydria, particularly if the general condition of the patient is good. At the same time even all these symptoms do not entitle us to make a positive diagnosis. The continuous secretion of gastric juice may cause similar symptoms, or there may be a simple hyperesthesia of the mucous lining of the stomach and gastritis, or even an ulcer. The diagnosis is rendered still more difficult if the symptoms are very slight; if they recur at irregular intervals; if the periods between the attacks are characterized by the complete absence of pain; and if the patient is afflicted with a variety of nervous, hysteric, or neurasthenic symptoms.

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[1] *Berlin. klin. Wochenschr.*, 1901, vol. xxxviii., p. 1077.

The diagnosis of hyperchlorhydria, therefore, can be made only from the examination of the stomach-contents. It will be found that the stomach-contents removed two or three hours after a test-meal consists of a very small quantity of a fine, well-digested, thin mass of food, containing a great deal of hydrochloric acid, particularly of free hydrochloric acid.

[In exceptional cases it is found that patients complain of symptoms of hyperchlorhydria, and the gastric contents shows a high acidity, which depends, however, upon combined chlorids rather than upon free HCl. Such cases are sometimes seen when there is no obstruction, and they are not fully explained.—Ed.]

Some time after meals or after longer periods of fasting the stomach will be found empty, whereas in cases of continuous secretion of gastric juice the stomach will always contain more or less material, consisting chiefly of gastric juice.

Another question—namely, whether the hyperchlorhydria is complicated by the retention of ingesta for an abnormally long period of time: in other words, by motor insufficiency—can also be decided alone by the examination of the stomach-contents.

The same applies to the question whether or not we are dealing with so-called acid gastritis. The decision in these cases will have to be rendered by the presence or absence of abundant quantities of mucus in the stomach-contents. The appearance, too, of numerous cell-nuclei is frequently significant; abundant quantities of mucus in cases of this kind can be aspirated from the stomach after fasting. Another question might arise—namely, whether or not hyperchlorhydria is complicated by gastroptosis or a vertical dislocation of the stomach, a condition that is particularly frequent in women. The only way to determine this is to examine directly the position of the stomach, using inflation and other adjuvants if necessary.

If it is determined that hyperchlorhydria is the cause of the symptoms, it must be decided what the significance of this hyperchlorhydria is—that is, whether we are dealing with a purely nervous form of the disease; whether there is some direct irritation of the mucous membrane of the stomach; or whether possibly there is an ulcer of the stomach. The diagnosis of round ulcer of the stomach is exceedingly easy in those cases in which the symptoms are well pronounced—that is, in which we find circumscribed areas of pain, regularly recurring attacks of cardialgia, vomiting of blood, and other so-called ulcer symptoms. In cases, however, in which these symptoms are not pronounced, the diagnosis may be exceedingly difficult. It is always a difficult matter to exclude ulcer, for we know that under certain circumstances ulceration of the gastric mucosa may exist and still produce no symptoms for a long time. If we see violent attacks of hematemesis combined with hyperchlorhydria, ulcer is probably present. On the other hand, we frequently see small hemorrhages in cases of simple hyperchlorhydria; these are the result of slight erosions of the mucous lining of the stomach. Circumscribed painful points are absent in many cases of ulcer, particularly if the lesion is situated on the posterior wall of the stomach. Attacks of cardialgia occur with greater regularity in ulcer than in hyperchlorhydria, but may also occur in the latter condition. If they are present in hyperchlorhydria, they may also occur at regular intervals, as in ulcer, and, on the other hand, they may be completely absent in ulcer. It is true that in the majority of cases a careful study of the whole symptom-complex will enable us to decide whether we are dealing with ulcer or simple hyperchlorhydria. It appears to me that one of the most decisive factors is that the symptoms in hyperchlorhydria are much more irregular than in ulcer. We must confess, however, that even if all these points are carefully considered, there are a number of cases in which a definite decision cannot be rendered.

Hyperchlorhydria may also be confounded with gall-stone colic. In the majority of cases, it is true, it is simply a matter of distinguishing an attack of gall-stones from one of the painful attacks that are caused by hyperchlorhydria. If it can be determined that the liver is acutely enlarged; that the gall-bladder is swollen and exceedingly painful to pressure; if icterus is present; if the attacks occur at irregular intervals and are far apart—the diagnosis is, of course, simple. There are, however, a great many cases in which the diagnosis is difficult. Hyperchlorhydria may run a latent course for a long time and cause violent paroxysms of pain only at long intervals; here, too, the pain may be chiefly located in the region of the gall-bladder or the region of the pylorus. Icterus, it is true, is always absent, but icterus may be absent in cholelithiasis. Pain in hyperchlorhydria usually appears at the height of digestion, whereas in gall-stone colic it may appear at any time and may be altogether independent of the time at which the meals are taken. If all these factors are carefully studied and the patient is observed for a long time, it will be an easy matter, as a rule, to make a differential diagnosis between these two diseases. If the patient, however, is seen during his first attack of pain, and if this attack for a long time is the only one, the diagnosis may be rendered exceedingly difficult, particularly as hyperchlorhydria may occasionally complicate cholelithiasis.

If the patient can be observed for a long time, the two diseases should not be confused with any of the diseases enumerated above. It is important to know that hyperchlorhydria is frequently overlooked for the reason that it produces so few and such slight symptoms. In order to protect ourselves against this oversight it will be well to aspirate the stomach-contents in every case that presents itself with mild dyspeptic symptoms, the exact origin of which cannot be determined. If the stomach-contents is carefully analyzed, it will be difficult to overlook hyperchlorhydria.

[Owing to an exaggerated sensitiveness to free hydrochloric acid, many patients present symptoms which closely resemble those of hyperchlorhydria when the amount of acid is not in excess; indeed, when it is even below the standard. Attention has been called to these by Sansoni,[1] Stockton,[2] and other observers. In this condition relief is had not so much in the decrease of acidity, as in the diminution of the hyperesthesia.—Ed.]

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[1] *Presse Medicale*, 1900.

[2] *Jour. Amer. Med. Assoc.*, January 11, 1902.

Treatment.—We have seen that hyperchlorhydria appears only at certain times—that is, when the stomach is irritated. The true nature of the disease is the increased secretion of hydrochloric acid at the time of digestion.

Our first object in the treatment of these cases must therefore be to avoid everything that leads to an increased glandular secretion in the stomach; in other words, a rational therapy of this disease, in the first place, must be prophylactic. It would lead us too far to discuss all those measures that might have to be employed were we to consider all the different factors that might influence this perversion or predispose to it. The measures to be employed will frequently depend on the nature of the primary disease, and will consequently vary greatly. If, for instance, hyperchlorhydria occurs after great mental exertion or after emotional excitement, it will be our task and our duty to advise the patient to avoid such excesses. If hyperchlorhydria is the result of an ulcer of the stomach or of chlorosis, treatment will have to be directed against these two diseases, and, at the same time, against hyperchlorhydria. In this place we will only discuss the treatment for hyperchlorhydria itself. The first postulate is a careful regulation of the diet.

Everything that is capable of increasing the secretion of hydrochloric acid should be carefully avoided. All food should be thoroughly masticated, so that it cannot irritate the stomach mechanically. Granular substances, nuts, fruit, pips, also sharp condiments, pepper, paprica, mustard, vinegar, radishes, horse-radish, strong alcoholic drinks, salads, and similar articles of food are to be interdicted. The patient should also avoid eating or drinking articles of food that are too hot or too cold. Coffee is to be absolutely interdicted. For breakfast the patient should take weak tea with milk or milk alone; cocoa, too, is generally well borne.

The most rational treatment for this condition appears to be the administration of large quantities of proteid, as hereby the excess of hydrochloric acid is combined; at the same time the ingestion of amylacea should be reduced. As a matter of fact, cases of this kind tolerate a diet containing much albumin very well, and seem to thrive on meat, eggs, and fish, whereas, on the other hand, carbohydrates, particularly if they are eaten in abundant quantities, are apt to cause disturbances. An objection has been formulated to this method of feeding the patients—namely, that albuminous material as such is capable of stimulating the secretion of gastric juice to a greater extent than carbohydrates. This statement, however, has never been positively verified; on the contrary, clinical observation seems to show that albuminous food agrees better with these patients than carbohydrate food. It must be remembered, of course, that the two are not equivalent in regard to their caloric value.

The power of combining hydrochloric acid varies in different articles of food. We are indebted particularly to Fleischer for some very exhaustive investigations in this direction. He has found, for instance, that veal, beef, mutton, raw ham, and Leube-Rosenthal's meat-solution are capable of binding twice as much hydrochloric acid as an equivalent quantity of calves' brains, calves' thymus, liver sausage, and similar articles of food; the former kinds of meat are, therefore, to be preferred in cases of hyperchlorhydria. 'It is improbable that those articles of food that bind more hydrochloric acid also cause a greater secretion of hydrochloric acid. This statement has been repeatedly made, but it is rendered improbable by the fact that the symptoms, instead of increasing after the ingestion of these articles, in reality decrease. Clinical observation, at all events, does not favor this view, but seems to teach us, on the contrary, that those articles of food that are capable of binding large quantities of hydrochloric acid are, as a rule, better borne than those that do not possess this property.

Fleischer designates the following articles of food as particularly suitable for cases of hyperchlorhydria, owing to their property of binding relatively large quantities of hydrochloric acid (in addition to those already enumerated): boiled ham, pork, and other articles of food, Swiss cheese, Roquefort, pea-sausage, pumpernickel, and gray bread; white bread is less suitable. He also recommends milk and cocoa. Patients with hyperchlorhydria should not be allowed to drink beer.

Owing to the fact that in cases of hyperchlorhydria free hydrochloric acid appears earlier in the stomach-contents than in normal cases, and that, consequently, the period of amylolytic digestion is abbreviated so that the conversion of starch into sugar can be carried only to a certain stage, relatively small quantities of amylaceous food should be given; it should, moreover, be finely distributed, and always be given in combination with abundant quantities of albuminous food, particularly meat. It is also well to replace starchy foods by articles of diet that have been in part dextrinized (Zwieback, toast, Kindermehl, maltoleguminose, baked bread, etc.). Particularly suitable articles of diet are the different flours that contain much proteid, as oatmeal, aleuronat flour, especially when these are administered as soups or mushes.

Whereas carbohydrates administered in the form of undissolved amylacea are capable of causing a great secretion of hydrochloric acid, they do not possess this property if they are administered in the form of sugars in solution. Strauss[1] has performed a number of experiments in this direction in my clinic, and has shown that if amylacea are introduced into the stomach in the latter form, they cause only a slight secretion of hydrochloric acid. On the strength of these investigations he recommends the administration of sugar solutions to such patients. Strauss repeatedly gave cases of hyperchlorhydria from 200 to 300 c.c. of 20 per cent. dextrose solutions *pro die*, and never saw any disturbances from this method of feeding. The administration of sugar is contraindicated in those cases in which there is motor insufficiency.

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[1] *Zeitschr. f. klin. Med.*, vol. xxix.

Potatoes should be given only in small quantities, mashed or boiled in bouillon. All vegetables like cabbage and turnips or salads are to be forbidden. Butter and cream are good, provided the patient does not dislike them.

The patient may be allowed to drink during meals; as a matter of fact, drinking large quantities of fluid helps dilute the gastric juice. The so-called acid mineral waters are particularly suitable; for instance, Apollinaris, Seltzer, Fachingen, Biliner Sauerbrunnen, and others. It is claimed that the carbonic-acid gas that is dissolved in these waters exercises a sedative effect, and that the alkaline carbonate that they contain helps neutralize the excess of hydrochloric acid. These waters can be given pure or with wine or milk.

[Strauss and Aldor[1] advise the liberal use of fats of various kinds in gastric hyperacidity, having in mind the necessity of the economy for a large number of calories of non-nitrogenous substances, which are not easily obtained from carbohydrates in the presence of the excess of hydrochloric acid in the gastric juice. This view has been corroborated by the experiments of Ewald, Boas, von Noorden, and others. Backman[2] recommends fats, especially cream, on the ground that they lessen the acidity, do not disturb the motility, and are usually well borne.

In practice this is found to be a useful addition to the diet, but occasionally fats cannot be given successfully. Sometimes sugar is well tolerated, and in many cases thoroughly cooked starchy food, especially when partly dextrinized, may be allowed in the early part of the meal.

Bialokur[3] advises that in hyperchlorhydria the diet, as far as possible, should be composed of carbohydrates to the exclusion of proteids, and he further advises that no sodium or chlorin should be administered as remedies.—Ed.]

It need hardly be mentioned that all food should be properly prepared; it should be as soft as possible, and no strong spices or condiments should be added.

These are the most important regulations in regard to the diet. All that has been said, however, applies only to cases of pure hyperchlorhydria; if atony or ulcer is present at the same time, the rules will be different. We will discuss the dietary regulations in these two diseases when discussing them in a separate section, and refer to these paragraphs for detailed information.

In conclusion I think it will be of value to my readers to inspect Fleischer's tables on the power of different kinds of meat and other articles of diet to bind hydrochloric acid. It may be found useful to consult these tables when arranging a diet-list for patients with hyperchlorhydria. One hundred gm. of pure egg-albumin bind 5 gm. of pure, 20 gm. of 25 per cent., hydrochloric acid, and 40 gm. of dilute muriatic acid (12.5 per cent.):

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[1] *Zeitschr. f. diat. u. physikal. Therap.*, vol. i., No. 2.
[2] *Zeitschr. f. klin. Med.*, vol. xlix., p. 249.
[3] *Gazeta Lakarska*, No. 4.

MEATS.			
100 grams.	Pure HCl	25 per cent. HCl.	Dilute muriatic acid.
Calves' brains, boiled	0.65	2.6	5.2
Liver-sausage .	0.80	3.2	6.4
Calves' thymus, boiled	0.90	3.6	7.2
"Mett" sausage	1.00	4.0	8.0
Cervelat sausage	1.10	4.4	8.8
Blood sausage .	1.30	5.2	10.4
Pork, boiled . .	1.60	6.4	12.8
Ham, boiled . .	1.80	7.2	14.4
Ham, raw . . .	1.90	7.6	15.2
Mutton, boiled .	1.90	7.6	15.2
Beef, boiled . .	2.00	8.0	16.0
Veal, boiled . .	2.20	8.8	17.6
Leube-Rosenthal meat solution	2.20	8.8	17.6

OTHER ARTICLES OF FOOD.			
100 grams.	Pure HCl	25 per cent. HCl.	Dilute muriatic acid.
Beer	0.10	0.40	0.80
Milk (an av. of different kinds)	0.36	1.44	2.80
White bread . .	0.30	1.20	2.40
Graham bread .	0.30	1.20	2.48
Black bread (gray bread) .	0.50	2.00	4.00
Pumpernickel .	0.70	2.80	5.60
"Hand" cheese	1.00	4.00	8.00
Fromage de Brie	1.30	5.20	10.40
Edam cheese . .	1.40	5.60	11.20
"Backstein" cheese	1.70	6.80	13.60
Pea sausage . .	1.70	6.80	13.60
Roquefort . . .	2.10	8.40	16.80
Swiss cheese . .	2.60	10.40	20.80
Cocoa	4.10	16.40	32.80

According to my experience, no fixed rules can be given in regard to the frequency with which the patient should eat. Bouveret advises three meals a day, and theoretically he is probably right, for, by allowing such long intervals to elapse between the time of eating, the stomach is allowed to rest. As the organ is abnormally irritable, such periods of quiescence are of value. In practice this regulation can probably be recommended in those cases that enjoy a good appetite. There are, however, a great many patients who can take only very little food at each meal, so that they have the desire to eat at frequent intervals; as a matter of fact, many of these patients feel better if they take a small quantity of food at frequent intervals.

I believe it will be an easy matter to arrange diet-lists with the aid of the rules that we have formulated and some of the tables that we have rendered in the first part of this work. In these tables the amount of proteid and carbohydrate contained in the different articles of diet is given. At the same time I think it advisable to insert a particular diet-list here. The table on page 321 is quoted from the cook-book of Biedert and Langermann, which we have frequently referred to.

However important the regulation of the diet may be in cases of hyperchlorhydria, this alone does not suffice; in many cases we are forced to use drugs, and among the remedies employed alkalis are the most important; if they are given in the right quantity and at the right time, they can certainly neutralize the acid of the stomach. They are indicated and permissible only in those cases where there is really an excess of acid. Many authorities have claimed that the administration of alkalis ultimately leads to an increased production of hydrochloric acid; this, however, is directly contradicted by practical experience. They undoubtedly act as irritants, and consequently cause a secretion of acid if they are introduced into the stomach when it is empty and contains no acid. No one will deny this, but to give these

DIET-LIST FOR PATIENTS WITH GASTRIC HYPERACIDITY.

	Grams albumin.	Grams fat.	Grams carbohydrate.	Calories.
A. In the morning, between 7 and 8 o'clock, 500 c.c. of milk, 40 gm. of toast, contain	20.3	18.4	55.8	483
B. In the morning, at 10 o'clock, 70 gm. of broiled veal (or 100 gm. of stewed veal without the skin, prepared as white ragout), or beefsteak, or fowl, 30 gm. of toast, 1 egg, 2 Zwieback (20 gm.), $\frac{1}{8}$ of a liter of wine, contain	32.3	12.0	36.9	395
C. Twelve o'clock at noon:				
(*a*) French soup with yolk of egg, contain	4.0	9.2	7.7	134
(*b*) 140 gm. of broiled or boiled fowl, roast meat, goulasch or haché, 200 gm. of raw meat, as beefsteak, or 140 gm. of finely chopped boiled beef or fish, contain	42.8	10.4	. . .	272
(*c*) Asparagus with cream gravy (a few heads of asparagus and half a spoonful of gravy), 20 gm. of toast, contain	2.2	1.2	16.4	87
(*d*) Omelet soufflé	12.1	18.3	9.6	259
(*e*) One small cup of black coffee[1]	. .	. .	. . .	. .
D. In the afternoon, at 4 o'clock, 250 gm. of milk-cocoa, 3 Zwieback	13.5	15.8	44.6	385
E. In the evening, at 7 o'clock:				
(*a*) 70 gm. of cold meat with 100 gm. of meat-jelly, 20 gm. of toast	24.2	5.4	15.4	212
(*b*) 20 gm. of Swiss or Dutch cheese	5.4	6.1	0.5	81
Total	156.8	96.8	136.9	2308

VARIATIONS.

	Grams albumin.	Grams fat.	Grams carbohydrate.	Calories.
A. In the morning, between 7 and 8 o'clock, instead of the above diet, 250 gm. of milk-cocoa, 2 eggs, 3 Zwieback	25.5	25.8	44.6	527
B. In the morning, at 10 o'clock, instead of the above diet, 70 gm. of ham, tongue, or corned beef, 1 egg, 30 gm. of toast	25.4	12.7	23.1	317
C. In the forenoon, at 10 o'clock, instead of the above diet, bouillon, scrambled eggs made from three eggs, 40 gm. of toast	22.3	20.6	32.3	415
D. In the afternoon, at 4 o'clock, instead of the above diet, 250 gm. of milk-cocoa, 1 egg, 2 Zwieback	18.3	20.1	37.7	416
E. In the evening, at 7 o'clock, instead of the above diet, 100 gm. of raw or scraped meat, as beefsteak, 20 gm. of toast	23.1	5.4	15.4	208

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[1] I think it advisable to omit black coffee.

remedies at this time is altogether useless. If no acid is present, it is unnecessary to give a neutralizing drug; in other words, the administration of alkalis in hyperchlorhydria when the stomach is empty is a direct error. If given in this disease, they should always be administered at the height of digestion. It is necessary to determine, by a series of tests, how much alkali is needed in each individual case. I have been in the habit of giving equal parts of bicarbonate of soda and burnt magnesia with sugar of milk; the dose I give varies from a quantity about as large as a pea to half or even a whole teaspoonful after eating. Some authors do not like to give bicarbonate of soda, because the administration of this drug causes the development of an abundant quantity of carbonic-acid gas. In cases of hyperchlorhydria in which atony is also present I concede that this objection is valid; I do not believe, however, that it applies to cases of simple hyperchlorhydria. Another objection that has been formulated is that the sodium chlorid that is formed in the stomach from sodium bicarbonate increases the secretion of gastric juice, but I do not think that this objection is valid, because the amount of sodium chlorid formed is very small. Jaworski recommends biborate of soda. If it is desired to give narcotic remedies together with antacids, 0.05 of codein may be added to a capsule containing 30 gm. of some antacid drug.

Other narcotic drugs are indicated only in cases of severe cardialgia. I think the best of these remedies are the preparations of belladonna (extract of belladonna, 0.03 in each dose, possibly together with 0.0005 to 0.001 of atropin). According to Penzoldt and others, these drugs aid in reducing the secretion of gastric juice. I hardly believe, however, that they should be applied for this purpose alone, for, as a rule, we administer narcotics only if pain is present; we rarely give them as prophylactics. At the time of the attack of pain, excessive secretion has already occurred, consequently the administration of these drugs for the purpose of checking secretion would be altogether irrational. Morphin should be given only in cases of extreme urgency.

In view of the fact that an abundant quantity of alkaline saliva is secreted during the mastication of the food, Bergmann[1] advises his patients with hyperchlorhydria to perform chewing movements during the whole period of digestion. He claims that the saliva secreted by this act binds the excessive acid of the stomach-contents, and reports excellent results in all cases of acid dyspepsia that were treated in this manner. Bergmann has prepared special chewing tablets for this purpose, to which he adds as adjuvants ginger-root or calamus-root for the purpose of stimulating the secretion of saliva, or burnt magnesia and ammoniomagnesian phosphate in equal proportions, in order to increase the alkalinity of the saliva. Theoretically, these chewing tablets may be rational; in reality the neutralizing action of the saliva is so insignificant as hardly to be worthy of consideration; the alkalinity of the saliva is much too slight to do any good in this respect.

Among the mineral waters the following are very popular: Carlsbad, Vichy, Neuenahr, and others. According to Jaworski,[1] the prolonged use of Carlsbad water decreases the secretion of gastric juice. Spitzer[2] also observed a reduction of superacid values to normal values for hydrochloric acid in the gastric juice after the prolonged administration of Carlsbad waters. The reports on the influence of saline waters on hyperchlorhydria are not uniform; some authors claim that sodium chlorid causes a reduction in the secretion of hydrochloric acid; others that it causes an increase.

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[1] *Berlin. klin. Wochenschr.*, 1895, No. 6.

The laxative action of the above-mentioned mineral waters is also of value in many of these cases, as there is frequently a tendency to constipation; at all events, practical experience teaches us that these waters are useful and assist in the cure of these cases. The exact manner in which they act is not altogether understood. The only conditions that contraindicate their administration are severe degrees of atony or ectasy.

In obstinate cases silver nitrate has been recommended. It is claimed that this drug can reduce the gastric secretion and the hypersensitiveness of the mucous membrane of the stomach. The silver salt can either be administered internally or it can be given as a stomach douche. If the stomach is irrigated in this way, the concentration of the solution should not be more than 1 : 1000. After the administration of a silver nitrate douche the stomach should be thoroughly washed with plain water.

Lavage of the stomach is usually unnecessary in cases of uncomplicated hyperchlorhydria; if atony exists at the same time, lavage is urgently called for. Occasionally this procedure is necessary in cases of simple hyperchlorhydria that are complicated with violent spasmodic attacks of pain. Even here, of course, the value of this treatment is only transitory, and, as a rule, we can get along very well without lavage, as the other methods mentioned above will usually suffice.

Electric treatment has also been recommended in the treatment of hyperchlorhydria. Whether any one has ever succeeded in permanently influencing the secretion of gastric juice by this treatment is exceedingly doubtful. The object of electric treatment in this case would, of course, be to reduce the secretion of gastric juice. If there is violent pain, internal galvanization can hardly be carried out; even the percutaneous method of applying electricity will rarely be employed, as the necessary apparatus is generally not at hand when the attack of pain begins. For all these reasons, this method of treatment need hardly be considered in practice.

The constipation that frequently exists can usually be treated with the above-named mineral waters, or possibly with injections or by massage of the abdomen. We need hardly mention that a great many other modifications of these different forms of treatment exist, and that many other methods have been devised for treating hyperchlorhydria. Much will depend on the cause of the hyperchlorhydria and the general health of the patient. All we wished to delineate in this section were those methods of treatment that are of value in the treatment of hyperchlorhydria itself.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxvii.; *Wien. med. Presse*, 1888.

[2] *Therapeut. Monatsh.*, April, 1894.

[It has been thought by Simon that the overacidity of gastric juice can be materially reduced by the employment of sweat-baths. His conclusions have been opposed by the investigations of Edel,[1] but in reply to the latter Simon[2] reiterates his statement, and claims that the effect is seen only in cases of superacidity, and that the baths have no effect on diminished or normal acidity. He also holds that the chlorids in general are reduced by means of free perspiration. Apparently there is some ground for the contention that vapor-baths lower high acidity of the gastric juice, but there seems to be some question as to whether the effect is long continued, and also as to the decrease in the chlorids of the body.—Ed.]

2. EXCESSIVE FLOW OF GASTRIC JUICE, GASTROSUCCORRHEA, CONTINUOUS SECRETION OF GASTRIC JUICE, HYPERSECRETION OR PARASECRETION.

Introduction.—Hypersecretion, parasecretion, excessive flow of gastric juice (Reichmann), continuous secretion of gastric juice (Riegel), is not a disease *sui generis*, but merely a perversion of function. In this condition the mucous lining of the stomach secretes large quantities of gastric juice, even when the stomach is empty and when there is no irritation of the mucosa from ingesta ; consequently gastric juice in abundant quantities will be found in the stomach even after the stomach has been at rest and no digestive irritation has occurred. We will refer below to the different factors that can produce this condition, and we will see how this abnormal secretion of gastric juice is produced : whether it is caused by nervous influence, by mechanical, chemical, or other irritants, or whether, as some authors claim, this continuous secretion of gastric juice is merely one of the features of gastrectasy. All authors seem to agree that this is a peculiar and a distinctly characterized perversion of function. Whosoever has seen a single typical case of this kind cannot deny that the disease-picture has certain characteristic traits. The general symptom-complex, it is true, may vary in many respects, just as in hyperchlorhydria. As in the latter condition, we find mild and severe forms of hypersecretion ; in the milder cases the symptoms produced by gastrosuccorrhea are so insignificant that they may easily be overlooked ; in the more severe cases the symptom-complex is so well characterized that a probable diagnosis of the condition can usually be made at once.

Reichmann deserves the credit for having first called attention to certain peculiarities of this condition and of the symptom-complex that it produces, and of having reported a case of this disease for the first time. I think it will be of interest to my readers if I briefly review Reichmann's observations in this case.

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[1] *Zeitschr. f. klin. Med.*, vol. xlii., Nos. 1, 2.

[2] *Ibid.*, vol. xlii., Nos. 3, 4.

The case was reported by Reichmann in 1882. The patient was a man of twenty-seven years who had been a sufferer from stomach-trouble for six years previous to the time when he began treatment with Reichmann. The patient claimed that his trouble originated from drinking large quantities of cold well-water. The first symptoms that he complained of were permanent pain in the pit of the stomach and vomiting after nearly every meal. This condition persisted for two years, and then the vomiting stopped, but the pain remained. The appetite was always good—if anything, increased. When the patient came to Reichmann the pain was his chief complaint. The character of the pain was spasmodic; the attacks began toward evening, lasted all night, and were most intense toward morning; at the same time there were heartburn and a great deal of thirst. No dilatation of the stomach could be determined.

The stomach was pumped out in the morning before any food was introduced, and about a liter of a dirty grass-green fluid was aspirated, containing undigested morsels of white bread and barley. This fluid gave distinct hydrochloric acid reactions. Its acidity was from 2 to 3 *pro mille.* The patient always felt better, the heartburn and the pain disappeared, as soon as the stomach was emptied.

In order to determine the origin of this fluid that was found in the morning when the stomach should have been empty the stomach was first carefully washed out in the evening, and the patient instructed to eat nothing and to drink nothing until the next morning; notwithstanding this a considerable quantity of fluid was found in the stomach the next morning in four distinct examinations. The stomach-contents consisted of an acid, almost transparent, fluid, of an average acidity of 2.5 *pro mille.* There were usually from 180 to 300 c.c. present. The fluid turned tropaölin brown and methyl-violet blue. Fibrin was peptonized by it within a short time.

Reichmann decided that the primary cause of all the disturbances observed in this case was a morbid increase in the secretion of gastric juice.

This attracted comparatively little attention at the time. Two years later Reichmann reported another analogous case.

In the following year—1885—Sahli and Schütz reported other cases. Sahli's case is particularly important because it constitutes the first observation of the intermittent form of this perversion. I also reported two typical cases of continuous secretion of gastric juice in the same year. In the first case I was able to aspirate some 500 c.c. of fluid at one time, and 420 c.c. at another, after the patient's stomach had been thoroughly cleansed the evening before and no food had been administered for twelve hours. In the second case the amount of secretion pumped out fluctuated between 70 and 100 c.c., even when the stomach should have been empty and after a prolonged period of fasting.

Jaworski, Gluzinski, von der Velden, Sticker, and others reported a number of interesting and very creditable observations at this time and shortly thereafter. In 1887 Honigmann was able to report 32 cases from my clinic alone, and Reichmann could report 16 cases that he observed himself. Since that time the reports on cases of this character have accumulated to such an extent that it is impossible to enumerate all the communications.

A few words in regard to the best name to give this perversion of function. Many authors nowadays still adhere to the original term, hypersecretion. It appears to me, however, that the true nature of the perversion is not expressed as precisely as it should be by this term: it seems to me that it is not sufficiently distinguished from hyperchlorhydria. The term that Reichmann proposed later on—flow of gastric juice, gastrosuccorrhea—seems to me more appropriate. I have proposed the term "continuous secretion of gastric juice" for the chronic forms of this disease, and intended to express by this term that this affection consists in a continuous secretion of gastric juice even though no food is introduced, and that in this respect it is different from hyperchlorhydria. This designation, it is true, applies only to the chronic forms of the disease, not to the intermittent forms, which we will presently discuss. Devic and Bouveret have proposed calling the disease Reichmann's disease. While I wish to express my full appreciation of the services of Reichmann in the discovery of this condition, I cannot refrain from remarking that the disease we are discussing is really no independent clinical entity, but merely a perversion of function that may either exist more or less independently or may be the result of other diseases.

Excessive secretion of gastric juice is seen in two forms—either as a transitory condition or as a persistent perversion. The former condition is called the intermittent, the latter the chronic, form. The intermittent form is frequently of nervous origin, but it can apparently also originate from other causes.

Many authors nowadays are inclined to consider the chronic form of excessive gastric secretion as a secretory neurosis. I have never been able to indorse this view. While I cannot deny that the disease may originate from some nervous trouble, it certainly can be caused by other factors. The continuous secretion of gastric juice is really due to a condition of permanent irritation of the mucous lining of the stomach, and is analogous in this respect to similar conditions that we see in other secretory organs. We know that certain nervous disorders lead to an increased and continuous secretion of tears, but we also know this excessive secretion of tears may occur secondarily as a result of numerous other diseases, particularly of inflammatory affections of the eyes. It appears to me that we have the same condition of affairs in the continuous secretion of gastric juice; the perversion may be a neurosis in many cases; however, it is undoubtedly in others the effect of some continuous irritation of the gastric mucosa, and in still other cases it is the result or a concomitant feature of a variety of diseases of the stomach. Jaworski and Gluzinski have selected still another term for this disease—namely, acid catarrh of the stomach. In a number of cases this designation seems to be happily chosen and correct; in others, again, it is hardly a suitable term.

In concluding these introductory remarks, I must call attention to the fact that Reichmann himself has emphasized that in this continuous secretion of gastric juice disturbances of the motor power of the stomach may also play a certain rôle. I mention this because many recent authors are inclined to attach the chief importance to this fact and to attribute the perversion primarily to motor disturbances.

(a) The Intermittent Form of Hypersecretion; Intermittent Secretion of Gastric Juice.—General Disease-picture and Symptomatology.—The name intermittent hypersecretion, which has been given to this form of the perversion we are discussing, designates that it is a secretory disturbance that occurs periodically. The symptoms appear in attacks that recur at irregular intervals. For this reason I believe that the term intermittent is better chosen than the other term that is sometimes used—namely, periodic.

Very little has so far been written on this intermittent form. This is probably due to the fact that the single attacks are of such short duration. Even though an attack may be accompanied by violent dyspeptic symptoms, it rarely lasts longer than one or a very few days. After the attack is over the patients recover completely and manifest no further symptoms.

For this reason the physician is frequently not even able to examine the vomit, and will very rarely have an opportunity of passing the stomach-tube in cases of this character. A careful observer, of course, will be struck by the fact that the patient vomits abundant quantities of an acid fluid periodically, and that this acid vomiting occurs even if the subject has eaten nothing for some time or has taken only a little water. At the same time it is impossible to determine whether the case is really one of intermittent gastrosuccorrhea unless the vomit can be examined by the physician himself; consequently the diagnosis acute dyspepsia or acute gastric catarrh, or if emotional disturbances precede the attack the diagnosis of nervous dyspepsia, is frequently made. Many patients state that they have frequently suffered attacks of this kind, and that the attacks seem to occur particularly after certain abuses—for instance, the excessive use of tobacco, after drinking very heavy wines, after emotional disturbances, etc. Many patients, moreover, do not call in a physician unless the attacks are exceptionally severe or are more protracted than usual.

The attacks themselves are characterized by the appearance of more or less severe pain in the region of the stomach, usually spasmodic in character. Vomiting occurs several times. The patients frequently remark that in the beginning particles of food are still found in the vomit, but that later on nothing is raised but a yellowish-green and acid fluid. Frequently considerable quantities of the latter are vomited.

An attack of this kind may last for several hours, for a day, or even for several days. As soon as the attack itself is over the patient feels perfectly well. Most of the sufferers from this disease enjoy a good appetite in the intervals between the attacks, and their general health is excellent; suddenly, after a short time—a few weeks or a few months—another attack occurs.

There are a number of cases in which the patients do not feel so well in the intervals between the attacks. Violent attacks of pain and profuse vomiting do not occur during these intervals, but the patients complain of a feeling of pressure in the region of the stomach, of general malaise, or of acid eructations after eating, particularly after the large midday meal. If cases of this character are carefully examined, it will frequently be found that the stomach does not perform its normal function, even in the intervals between the attacks. If the stomach is washed out at the height of digestion and the gastric contents analyzed, varying degrees of hyperchlorhydria will be found; at the time of the attack this hyperchlorhydria becomes excessive and constitutes a real gastrosuccorrhea. The same is found at the height of the attacks. The material that is vomited at the beginning of the attack contains morsels of food and much free hydrochloric acid. The values for hydrochloric acid in the material that is vomited later, and which does not contain morsels of food, are usually smaller. There are also a number of transitional stages between the intermittent form of gastrosuccorrhea and the chronic form, or the reverse may be the case—that is, the chronic continuous secretion of gastric juice associated with hyperchlorhydria may gradually improve or even disappear. From time to time, however, when the stomach is very much irritated, a transitory increase in the secretion of gastric juice may be observed, even without any stimulation by ingesta. In these cases hyperchlorhydria usually persists after the gastrosuccorrhea has disappeared.

We will now enter into a careful discussion of the different symptoms of this condition. The disease-picture will naturally be different if the patients are examined during the intervals between the attacks, than if they are examined during an attack. Patients who are sufferers from the intermittent form of gastrosuccorrhea usually feel well in the periods between the paroxysms; their general appearance is good and they enjoy a good appetite; in fact, nothing points to the existence of any perversion of gastric function. If the stomach is washed out during these periods of quiescence and after prolonged fasting, it will be found that the organ is completely empty.

If the stomach is pumped out at the height of digestion, it will also be found that the gastric juice is normal. There are, however, a number of cases that complain of mild dyspeptic disturbances in the interim between the attacks; cases of this kind are quite frequently seen; we have already mentioned them above. These persons complain of a feeling of slight oppression in the region of the stomach, a mild degree of discomfort, and suffer from sour eructations after eating. If the stomach-contents is aspirated at this time and analyzed, it will be found that a more or less pronounced degree of hyperchlorhydria, exists; in fact, a considerable degree of hyperchlorhydria may be found, and at the same time the patient be free from all subjective symptoms. Cases of this kind usually enjoy a very good appetite.

If the patients are examined during the attacks, the picture is altogether different. The attacks usually begin suddenly and unexpectedly: the patients awake in the middle of the night, most frequently toward morning—the attacks rarely begin during the day; the region of the stomach is very painful, there is a severe degree of heartburn and of acid belching; the pain rapidly increases in severity, and ultimately becomes spasmodic; then vomiting occurs, and very acid masses are raised. The first portions vomited usually contain particles of food; after a short period of rest vomiting recurs. This second vomit is usually yellowish or yellowish-green, and contains no particles of food whatever. If this fluid is carefully analyzed, it will be found to consist of gastric juice; it contains free hydrochloric acid in moderate quantity and possesses good peptic powers. Microscopic examination reveals the presence of isolated desquamated epithelia and of cell-nuclei. Occasionally a little mucus is found in this fluid, but usually in very small quantities; in fact, the amount of mucus is, as a rule, no greater than that which can be pumped out of a normal stomach when it contains no food. At times the gastric secretion will be found to be yellowish-green, from the admixture of a small quantity of bile constituents. The acidity of the gastric juice is, as a rule, moderately high—30 to 50; only the material that is vomited in the beginning—namely, that vomit which still contains particles of food—is hyperacid. The amount of vomit may vary greatly and may amount to several hundred cubic centimeters. Occasionally, though rarely, the vomit contains a little blood. Even if this is seen we are, of course, not justified in concluding that an ulcer must be present.

During the attacks the appetite is completely lost; the thirst, on the other hand, is much increased. Drinking some water usually relieves the patient a little; this relief, however, is only temporary, for the symptoms soon begin to exacerbate again, and after a short time the patient vomits. The patients themselves sometimes notice how large the quantity of vomited fluid is, and state that it is disproportionate to the amount of fluid previously swallowed.

There are great variations in the intensity and the duration of these attacks. In certain cases the pain is very slight and moderate; in other cases the pain may be very severe. The patients complain bitterly and moan; they throw themselves about on the bed; they grow pale, and their features show that they are suffering violent distress. After each attack of vomiting the patients feel relieved for a little time; pain, however, soon recurs and increases in severity until vomiting occurs again and a large amount of acid fluid is raised.

The pulse is usually accelerated and small during the attack; the skin is moist, and the face frequently covered with cold perspiration. The urine that is voided in the beginning of the attack is very scanty, dark, of a high specific gravity, and usually of an alkaline reaction.

An attack of this kind sometimes terminates suddenly. The patients claim to feel distinctly how the spasm of the stomach relaxes. In other cases again the pain and the spasm stop slowly and gradually.

After the attack the patients feel weak; they soon, however, relapse into sleep, and when they wake up, they often feel perfectly comfortable.

We have already stated that the appetite is altogether lost when the attack begins; the day after the attack, however, the appetite may be completely normal or even increased.

Occasionally the duration and the intensity of an attack may vary in individual cases: sometimes an attack will be very short; in other cases it will continue through several days. The general health of the patient will be affected in different ways: in some instances it will hardly be disturbed; in other cases it will be disturbed to a great degree.

In many cases of this character headache is an important symptom in addition to the gastric symptoms. This was the case, for instance, in those cases that were suffering from the symptom-complex that Rossbach grouped under the name of nervous gastroxynsis. It appears to me that we are hardly justified in classifying these cases separately simply because violent headache existed in addition to cardialgic attacks and vomiting of hyperacid material. Rossbach himself has correctly interpreted the connection between the two groups of symptoms, and has recognized that the presence of an abnormally large quantity of hyperacid material in the stomach is the true cause of the attacks that he calls gastroxynsis. His cases occurred in persons who were very nervous and who had overexerted themselves mentally. The fact that such a symptom as headache was very conspicuous in these cases need not surprise us; at all events, the occurrence of headache does not permit us to change our views as to the nature of this disease. Rossbach's description, it is true, does not enable us to determine positively whether the cases he reports were cases of hyperchlorhydria or cases of gastrosuccorrhea. At the time when Rossbach published his observations, no reports on gastrosuccorrhea or hyperchlorhydria had been published. Our views to-day are slightly different from those that prevailed at that time, and to judge from our personal experience, we feel justified in declaring that the disease described by Rossbach belongs to the perversion we are discussing and is not a separate perversion of function.

Etiology.—We know very little that is positive about the causes of this intermittent form. As in the case of hyperchlorhydria, we observe a tendency to gastrosuccorrhea most commonly in young, nervous, very active, and excitable persons; less frequently in old people. Hyperchlorhydria, it is true, occurs much oftener than intermittent gastrosuccorrhea; at the same time both perversions of function, as we have already mentioned, may occur simultaneously. If the attack occurs when the stomach contains food, the material vomited at first, as a rule, contains an excessive amount of hydrochloric acid. The material vomited later contains no particles of food and consists of pure gastric juice frequently mixed with a little bile; this second vomit is usually less acid.

Psychic excitement, mental overexertion, and similar states may be the direct cause of an attack of this kind; or, again, certain noxious agencies that affect the mucous lining of the stomach itself may start an attack.

In some people the attack begins after the ingestion of certain articles of food or when the stomach is overloaded. In other cases a drink of cold water or smoking a very strong cigar may precipitate the attack. Some investigators claim to see a connection between these attacks of intermittent gastrosuccorrhea and the gastric crises of locomotor ataxia. We fail to see a direct connection between these gastric crises and intermittent gastrosuccorrhea; if they were directly interdependent on each other, they would more frequently occur together. It is true that Sahli reported a case in which the two symptom-complexes seemed to run parallel. In other cases, however,—and I may say in the majority of cases,—of gastric crises gastrosuccorrhea is not observed. I have paid attention to this point, and agree with many other authors in denying any connection between the two in the majority of cases. If a large number of cases of this kind are examined, it will be found that the secretion of gastric juice varies greatly during the crises. The degree of hydrochloric-acid acidity, furthermore, does not vary greatly in the two conditions. All that we can say so far is that gastrosuccorrhea is observed in some cases of gastric crises. Possibly this can be explained by assuming that the nerve-tracts that are concerned in hydrochloric-acid secretion are abnormally irritated.

Diagnosis.—The diagnosis is easy if it is possible to perform a careful analysis of the vomit. It is true that the attack *per se* is distinguished by certain characteristic features, but it is easy to overlook those symptoms that may be considered typical for this condition; as a matter of fact, the diagnosis is rarely made from the clinical picture alone. The symptoms that would characterize an attack as one of intermittent gastrosuccorrhea would be repeated vomiting of large amounts of fluid at a time when no ingesta are present in the stomach.

At the same time a direct analysis of the vomit is essential in order to arrive at a definite diagnosis. If the above-named symptoms are observed, the disturbances that we are discussing may be suspected, particularly if it is found that vomiting or the administration of alkalis seems to relieve the symptoms. The only examination, however, that really decides the diagnosis is the analysis of the vomit—if this shows that the material raised possesses all the properties of gastric juice, that it contains hydrochloric acid and pepsin, and that its specific gravity is smaller than the mixture of gastric juice and food-particles that is raised at the height of digestion.

If the existence of gastrosuccorrhea has been established, it remains to determine whether or not we are dealing with the intermittent form. This question is readily answered; the symptoms intermit, and this clinches the diagnosis. If the stomach is pumped out in the interval between attacks or after a period of fasting, the stomach should be found empty.

The only way in which to decide whether this perversion of function is one of the symptoms of tabes or whether it is caused by some other disease is to make a careful examination of all the organs of the body and carefully to consider the anamnesis of the case before rendering a decision.

Therapy.—Treatment should be instituted in two directions: on the one hand, the attack should be treated; on the other, the primary cause of the disease should, if possible, be removed during the intervals between the attacks. In regard to the first task,—the treatment of the individual attacks,—the most rational procedure, without doubt, is to wash out the stomach thoroughly as soon as the attack begins. This lavage should be followed by irrigation with some alkaline fluid or with a 1 : 1000 solution of silver nitrate; if necessary, this procedure should be repeated. If lavage cannot be performed, large doses of some neutralizing remedy should be administered. To judge from my experience in the treatment of these cases, vomiting is not stopped by these measures; all that is accomplished is to quiet the patient for a time. A hard-boiled egg or a little milk if taken early during the attack will frequently mitigate the distress. If the pain is very severe, an injection of morphin with a little atropin should be given; cocain has also been recommended for internal use. The patient should be advised against drinking too much, because the ingestion of large quantities of fluid seems to favor vomiting; if the thirst is very severe, the patient should swallow small pieces of ice, or rectal injections of water or of salt solution should be given. The best treatment of all, however, is the introduction of the stomach-tube and thorough cleansing of the stomach whenever this is possible.

In regard to the second task,—the treatment of the case in the interval between the attacks,—the chief indication is to find the primary cause of the disease and to attack it directly. The first thing to determine in every case is whether or not the stomach performs its functions in a normal manner in the intervals between the attacks; it is not sufficient to determine that the stomach contains no gastric juice after a period of fasting; in other words, that gastrosuccorrhea is only intermittent. It is absolutely necessary, as in every other case of disease of the stomach, to determine the exact relation between the secretory and motor powers of the stomach during digestion. The stomach-contents should be examined at the height of digestion. It is particularly important to determine whether or not hyperchlorhydria exists in the interim; as I stated above, hyperchlorhydria of this character may be present and still run a latent course. If the presence of hyperchlorhydria can be determined, this perversion should be treated and remedied. In regard to the details of this treatment, I refer to the section on Hyperchlorhydria.

If it can be shown that the attacks originate from excesses in smoking, tobacco should be interdicted. If it can be shown that mental overexertion is the primary cause of the attack, the patient should be instructed to limit his mental labors and grant himself periods of rest. If the attacks originate from excesses in eating and drinking, the diet should be carefully regulated. In neuropathic subjects, in patients with neurasthenia, these conditions should be treated. In many of these cases a carefully supervised cold-water cure may be advantageous. It is impossible to formulate any general rules in regard to the treatment of these conditions. We must, unfortunately, concede that many cases baffle all treatment; the most careful examination and the most careful consideration of all the factors in the case may not enable us, after all, to prevent the recurrence of attacks or to remove the primary cause of the disease.

(b) Chronic Continuous Secretion of Gastric Juice; Chronic Gastrosuccorrhea; Chronic Hypersecretion; Chronic Parasecretion.—Nature and Etiology.—As I have already stated in the introduction, the chronic continuous secretion of gastric juice is not a disease *sui generis*, but only a perversion of function like acute gastrosuccorrhea, intermittent hypersecretion, or hyperchlorhydria. The functional disturbance is manifested by the secretion of an excessive quantity of gastric juice; this occurs not only when the mucous lining of the stomach is irritated by ingesta, but also after the ingesta has been removed from the stomach—that is, when the stomach is empty. This form of continuous secretion of gastric juice is, therefore, clinically characterized by the appearance of gastric juice in the stomach after fasting and when the organ contains no food.

Many investigators have formulated the objection against this definition, that too much importance is placed on the appearance of gastric juice in the stomach after fasting; they claim that it is due to this misplaced emphasis that a number of authors frequently report cases of gastrosuccorrhea, while others consider this condition a clinical rarity. They claim that gastrosuccorrhea is present only in those cases in which the whole symptom-complex that Reichmann first described is present, and that only in those cases in which we find this whole syndrome are we justified in speaking of "continuous secretion of gastric juice."

It appears to me that it is not wise to circumscribe our conception of the continuous secretion of gastric juice in this manner. The disease is characterized by the fact that large quantities of hydrochloric acid are secreted by the stomach after fasting. As a rule, this abnormal secretory action of the stomach causes certain distressing symptoms; if this were not the case, the patients would not consult a physician: at the same time none of these symptoms can be called pathognomonic, nor does the whole symptom-complex necessarily prove that there is a continuous secretion of gastric juice, even though the peculiarities of the disease-picture may lead a physician who has seen many such cases to make this diagnosis.

I might compare the difficulties encountered in regard to the interpretation of this stomach symptom with those of another met with in practice—namely, albuminuria. There are cases in which the whole symptom-complex of nephritis, of albuminuria, is present,—viz., the pulse of high tension, acute hydrops with edema in characteristic parts of the body, etc.,—so that nephritis or albuminuria can readily be suspected. In other cases, again, albuminuria is discovered by chance, or rather is discovered only by those physicians who look for it carefully. Although in the latter instance all other symptoms that point to disease of the kidney are absent, we still speak of albuminuria just as we do in the former case.

In the same manner, therefore, as we speak of albuminuria whenever we find albumin in the urine, even though no other symptoms are present, we speak of hypersecretion or continuous secretion of gastric juice in those cases in which large quantities of gastric juice are found in the stomach for a long period and at a time when the organ contains no food. Whether or not other serious or insignificant symptoms appear at the same time, we draw the conclusion from the appearance of an abundant quantity of gastric juice at an abnormal time that the function of the stomach is perverted, in the sense, namely, that the mucous membrane secretes gastric juice even without stimulation by ingesta. The exact cause of this perversion of gastric secretion will have to be determined in each case. I must emphasize, again, that the continuous secretion of gastric juice is primarily a perversion of function like atony of the stomach, like hyperchlorhydria, like albuminuria, like mellituria. In some cases of this disease, however, the continuous secretion of gastric juice is the most important symptom, so that the impression is created that excessive secretion of gastric juice is a disease *sui generis.*

It is true that a characteristic and peculiar clinical picture is frequently presented in cases of continuous secretion of gastric juice. When such symptoms are seen by an experienced physician, he will at once investigate whether an abundant quantity of gastric juice is present in the stomach, even though no food has entered the organ for some time. The clinical picture, however, may be very misleading; at least I myself confess to having frequently been misled. I have at different times felt justified in assuming the presence of continuous secretion of gastric juice from the general symptom-complex presented, and have been disappointed, on careful examination, to find that I was mistaken. In cases of this kind nobody can possibly speak of continuous secretion of gastric juice, even though the symptom-complex resembles the one that Reichmann first described, for, as a matter of fact, there is no continuous secretion of gastric juice. Inversely, however, I am of the opinion that we must speak of continuous secretion of gastric juice in those cases where we find abundant gastric juice in an empty stomach, even though all those symptoms are absent that we are accustomed to find in the majority of other similar cases; at the same time, we are justified in anticipating the occurrence of certain sequelæ, provided the continuous secretion of gastric juice persists. The continuous secretion of gastric juice is at best only a symptom; in some cases it appears to exist alone; in other cases it appears to be a secondary symptom, and in still other cases it appears to be the primary cause of a variety of secondary phenomena.

An objection that at first sight seems to be much more valid is the following: It may be that a certain secretion of gastric juice always occurs in a normal subject after fasting. It is true that this view would be in direct opposition to the opinions entertained by physiologists and clinicians for many years ; but, as a matter of fact, many authors have recently performed a number of investigations on the basis of which they claim that a certain amount of gastric juice can be aspirated from the stomach of healthy normal human beings even after a short period of fasting and when the stomach contains no food.

I cannot say that I am astonished at this statement. In the first publication on hypersecretion that I issued some ten years ago I called attention to the fact that we may expect to encounter cases in which a decision will have to be rendered whether or not the secretion of gastric juice is really pathologic. At that time I showed that a great many intermediary stages may be encountered between normal degrees of gastric secretion and an abnormal increase of this function.

Nobody will deny that under certain conditions a few cubic centimeters of gastric juice can be pumped out of the stomach of a healthy subject ; this fact is known to every physiologist. If the mucous membrane of the stomach in a dog with a gastric fistula is irritated with a glass rod, this simple contact will immediately cause a slight secretion of gastric juice ; in one animal we will obtain a few drops of secretion, in another an abundant quantity. We must assume that the same conditions exist in human beings. It is possible that saliva or mucus from the pharynx or other normal products that reach the stomach may irritate the mucous membrane sufficiently to cause a secretion of gastric juice. This, in fact, justifies the statement that normally a certain amount of gastric juice can be pumped out of the stomach of a healthy subject ; at the same time this does not justify us in speaking of a continuous secretion of gastric juice under physiologic conditions. As a matter of fact, numerous experiments by different authors have shown that large quantities of gastric juice are rarely found in the stomach after fasting ; if I say large, I mean quantities greater than 20 c.c. In the majority of cases nothing can be found, or, at best, from 1 to 10 c.c. I will not enter into a discussion of the question whether or not this secretion of gastric juice was possibly due to the irritation by the sound or to some other factor. The irritation of the sound can exercise an influence in this direction under certain conditions. This is demonstrated by an observation that I have frequently made in individuals who are not accustomed to the passage of the sound—viz. that more gastric juice is poured out in the beginning than later, when they are accustomed to the instrument. Whatever the true connection between the introduction of the sound and the secretion of gastric juice may be, a summary of all the observations in this respect shows that in only 6.4 per cent. of the examinations the results were positive in the sense that more than 20 c.c. were found in the stomach. I consider it at least doubtful whether the same result would have been obtained if this experiment had been repeated several times in the same individual. In order that we may speak of the continuous secretion of gastric juice we must not only find a certain quantity of the fluid, but the fluid must be more or less constantly present. An isolated experiment in a healthy subject that gives a positive result shows us that under certain circumstances the stomach of a healthy individual may contain a little gastric juice after the sound has been introduced, but it does not prove by any means that the stomach of a healthy individual continuously secretes gastric juice. We can speak of the continuous secretion of gastric juice only if considerable quantities of gastric fluid can be aspirated from the stomach of a healthy person who has fasted for some time; this gastric juice must be constantly found, and must be present at a time when the stomach contains no food. There may be a difference of opinion in regard to the amount of gastric juice that should be present in order to justify us in speaking of an abnormal secretion; some have declared that 50 c.c. must be present; others, 100 c.c. If more than 50 c.c. are constantly found in the stomach of a subject that has been fasting for some time and that has been accustomed to the introduction of the sound, I should certainly call this finding pathologic; at least I can recall no case in which the subject was perfectly normal and in which there were no disturbances in the sphere of the digestive organs, and in which, nevertheless, such quantities of gastric juice could be discovered after fasting; but even if such a case should occur, I should still be inclined to call it pathologic, even though all subjective symptoms were absent. There are a great many people who are afflicted with a mild degree of mellituria; in fact, there are many subjects who suffer from a quite severe degree of mellituria and still are free from all subjective symptoms. How often has a discovery of this kind been made by chance when the urine of some member of the family was examined for sugar because sugar had been found in the urine of other members of the same family. Mellituria has quite frequently been found under these conditions in subjects that were apparently in perfectly good health. Are we justified, in a case of this kind, in saying that mellituria is physiologic because all morbid symptoms are absent? Certainly not; and it seems to me that the presence of an abundant quantity of gastric juice in a healthy subject after a period of fasting is pathologic even if the patients complain of no particular symptoms. We are certainly not justified in calling such a phenomenon normal, particularly if this secretion of gastric juice is constantly found. It may be that in certain regions, where the inhabitants eat very irritating food, or where the diet consists largely of salted and smoked meats, small quantities of gastric juice containing free hydrochloric acid may frequently be found in the stomach of healthy persons after fasting. My experience with all those cases that have come under my personal observation has been that, as a rule, nothing is present in the stomach of a healthy subject after fasting, or at best a few drops or a few cubic centimeters of secretion.

It is true that the symptoms we encounter in cases of continuous secretion of gastric juice may vary greatly: they may be slight or very severe. It is true that they do not in any way run parallel to the intensity of the gastric secretion, nor are the symptoms more severe if the quantity of gastric juice that is secreted after fasting increases.

We have already shown, in discussing hyperchlorhydria, that a difference exists in different individuals in regard to the sensibility of the gastric mucosa to certain irritants; we may, therefore, encounter severe symptoms in cases in which the secretion of gastric juice is not great, and, inversely, we may encounter slight subjective symptoms in cases in which the secretion of gastric juice is continuous and abundant. The general symptom-complex, of course, will vary if there is stenosis of the pylorus, ectasy, atonic ectasy, ulcer, or some other complicating lesions.

Statements in regard to the frequency of this affection vary greatly; some authors claim that the disease is a common one; others that it is very rare. Honigmann, in 1887, reported 32 cases from my clinic, all cases that entered our wards in the course of only a few years. I think that none of these cases was doubtful; they were all cases of chronic hypersecretion of the type first described by Reichmann. Since that time I have observed a large number of cases myself. I will not formulate any statistics in this place, for they would not teach us anything in regard to the frequency of continuous secretion of gastric juice as compared with other diseases and perversions of functions of the stomach. The clinical material that a clinician sees is more or less uniform; and hospital physicians, as a rule, treat old cases. It may be that in other regions—for instance, in north Germany—the affection is less frequently encountered than in middle Germany. There is only one thing that I wish to emphasize, and that is that there was not a single case among my patients with hypersecretion, including my private patients,—at least as far as the statements of the patients themselves allowed me to formulate a judgment,—whose stomach-contents had ever been examined after a period of fasting and after a preliminary cleansing of the stomach, before they came into my care. Without such an analysis the question whether or not the stomach secretes gastric juice without being irritated by ingesta—that is, whether or not a true continuous secretion of gastric juice exists—cannot be decided.

Very little that is positive is known in regard to the causes of continuous secretion of gastric juice. I have been unable to determine that certain classes of the population are particularly predisposed to the disease; it is encountered in poor and rich alike, more frequently in men than in women, and more frequently in youth and in the middle years of life than in old age. If it is occasionally encountered in the aged, it will be found, as a rule, that it began long ago. I have been able to determine that the disease occurs more frequently in persons who are subjected to severe mental strain than in the ordinary laboring population.

The same noxious agencies have been made responsible for the occurrence of continuous secretion of gastric juice as for hyperchlorhydria—namely, rapid eating, insufficient mastication, the abuse of food that is hard to digest or is very irritating or contains spices and condiments in large quantities.

Psychic excitation has also been accused of causing the disease;

there is no doubt that the latter state predisposes to acute gastrosuccorrhea. Whether or not chronic continuous secretion of gastric juice can develop on this basis remains an open question.

Some authors claim that ectasy of the stomach may cause continuous secretion of gastric juice; that the two are related in some way is shown from the frequent occurrence of both together; what the exact connection is cannot, however, be determined by clinical observation. I will refer to this question again when discussing the symptoms of this disease.

Symptoms.—The symptom-complex of continuous secretion of gastric juice in pronounced cases and in cases of high degree has certain peculiarities that lead an experienced observer to suspect the existence of this perversion of secretion. In milder cases, however, the symptoms are frequently so little pronounced that there is great danger of overlooking the condition.

The majority of cases that come for treatment complain of certain gastric disturbances, and state that these have existed for some time, usually for many years; most patients state that the gastric symptoms began gradually; that they were only slight in the beginning; that they stopped after a time, and gradually returned with greater severity. In exceptional cases the patients claim that the gastric symptoms began suddenly or that they appeared acutely after some violent excitement, after some indiscretion in diet, after drinking cold water, or some other factor of this kind, and that they persisted ever afterward. In the beginning there are simply mild dyspeptic disturbances appearing some time after eating; these are manifested by a feeling of pressure and fulness and by sour eructations. These symptoms persist for a short time and gradually disappear. They generally appear after the midday meal at the height of digestion, and resemble in general the symptoms that we see in all cases of hyperchlorhydria. Gradually they begin to increase in severity; during the first few hours after an abundant meal the patient is usually free from pain; some hours afterward, however, pain appears, slowly growing in intensity and frequently assuming a spasmodic character; finally, vomiting occurs.

In contradistinction to simple hyperchlorhydria and other diseases of the stomach, the pain occasionally occurs before eating, and as soon as the patients eat something it stops. Another fact that can be learned from the anamnesis of these cases is that attacks of pain frequently begin at night—as a rule, between 11 and 2 o'clock; here, too, a small amount of food will, as a rule, relieve to a certain extent.

Quite frequently it is reported by the patient that all the symptoms existed for a time and then disappeared, only, however, to reappear again after the lapse of a certain time. A change in the occurrence of the symptoms is frequently encountered in the history of these patients. Finally, the symptoms grow more obstinate and more persistent, and the appetite, which had remained good or was increased for a long time, gradually disappears; vomiting is a rare occurrence in the beginning, recurs with greater frequency, and the patients gradually emaciate more and more. Finally, there may even be such a loss of strength that an inexperienced observer may be lead to make the diagnosis of carcinoma.

We will now discuss the individual symptoms. The most important of the subjective symptoms is pain; this is rarely absent in pronounced cases of continuous secretion of gastric juice. The attacks of pain occur at two different periods of time: first, during the period of digestion; second, when the stomach is empty. Immediately after eating the patient does not complain of pain; in fact, if pain was present before, it has a tendency to disappear when something is eaten. The period in which the patient is free from pain usually lasts only for a certain time; after several hours—the exact time varies—pain sets in; in the beginning it is slight, but it gradually increases in intensity, and finally may become very severe. In many cases vomiting occurs and seems to relieve the pain for a time.

The nocturnal attacks of pain are, to a certain extent, characteristic of this condition; the patients are awakened by disagreeable sensations, by a feeling of unrest and burning in the epigastrium. These sensations gradually increase in severity until vomiting occurs. The vomit usually tastes acid, occasionally bitter, and, as a rule, consists of more or less abundant quantities of a slightly cloudy fluid. On careful examination it will be found that the vomit consists either of pure gastric juice or of gastric juice containing a few remnants of amylaceous material.

If patients of this kind eat something, particularly some albuminous food,—an egg, a little milk, or some similar article of diet,—as soon as the pain begins, the distress may frequently be modified.

All these symptoms may recur daily or nearly every day for weeks or months, then they may stop for a long time, only to recur at some future time without any demonstrable cause; in other cases again they persist without interruption and simply vary in intensity. In severe cases of this kind vomiting may occur several times during the day.

The appearance of the patients varies according to the duration and the intensity of the disease, according to the existence or non-existence of other complications. All cases that have suffered from this disease for some time are more or less emaciated. The skin is usually dry and wilted, as in cases of severe diabetes, so that it can be raised in folds. The subcutaneous adipose tissue is almost gone; the muscles are flabby and atrophic; the patients appear very much reduced. Even an experienced observer may commit the mistake of diagnosing carcinoma in these cases, and only a careful examination may reveal his error; in fact, physical examination in these cases may seem to support the diagnosis of carcinoma, for we quite frequently find pronounced ectasy, or under certain conditions an abnormal resistance, in the region of the pylorus. Another factor that may speak in favor of carcinoma is a history of bloody vomiting, as this occurs in quite a number of cases. I have frequently been called in consultation to cases of this kind in which the diagnosis of carcinoma had been made from the symptoms enumerated above; only a very careful examination revealed that the disease consisted in a continuous secretion of gastric juice with ectasy of the stomach and cicatricial stenosis of the pylorus. The only way in which to arrive at a positive decision is to analyze the stomach-contents, particularly after a period of prolonged fasting. Cases, of course, are rare in which all the symptoms, as cachexia, extreme emaciation, loss of strength, ectasy, vomiting of blood, tumor of the pylorus, are found at the same time; but they do occur, and we should always be very careful in rendering a decision in regard to the cause of the trouble. They teach us that we should never construct a diagnosis on the basis of a congeries of symptoms that indicate a certain condition; a single symptom that does not fit into the frame should lead us to regard the diagnosis doubtful.

I may add that cases of this kind are very gratifying to the physician, for I have repeatedly succeeded in restoring such patients to almost complete health within a few weeks, and in causing an increase in weight of from 15 to 20 pounds.

The tongue presents no characteristic features in this disease; it frequently looks perfectly normal, is moist and red, and is not coated. In other cases, however, that are very severe or that are of very long duration, the tongue may be coated with a large or small quantity of thick mucus.

The teeth are frequently carious or defective. Possibly the frequent vomiting of very acid masses is responsible for this, or that decay of the teeth is caused by abnormal decomposition processes that occur in the mouth.

The appetite varies: it is more common to find the appetite good than the reverse; in many cases it may even be increased. The patients manifest a desire to eat at frequent intervals, and this desire may become so intense that they frequently develop canine hunger. Many patients themselves observe that the nocturnal attacks of pain are most easily remedied if a small quantity of albuminous food, either some hard-boiled and finely distributed white of egg or something else of that kind, is eaten as soon as the attack of pain begins. Sometimes the patients manifest a desire to eat again within a few hours after a relatively large meal. The same occurs after violent attacks of vomiting.

On the other hand, there are many patients whose appetite is increased, or at least good, but who are afraid to eat large quantities of food for fear of the pain and vomiting that they know will ensue.

A pronounced decrease of appetite or a positive disgust for food is only rarely encountered; the aversion for food usually applies to meat particularly. The same perversions of taste exist here, therefore, as in carcinoma. If the appetite, as in the majority of cases, is good or even increased, the patients, as a rule, prefer an albuminous diet, chiefly meat, eggs, milk, etc.; it is possible that they may notice themselves that food of this kind agrees better than a diet consisting chiefly of amylaceous material. There are, however, a number of exceptions to this rule.

The thirst is generally increased in the same way as the appetite;

occasionally the history of the patient shows that the appetite and the thirst are both morbidly increased to such an extent that, combined with the emaciation of the patient, a diagnosis of diabetes seems the most probable one. Thirst is particularly apparent in those cases in which a considerable degree of ectasy of the stomach exists in addition to severe continuous secretion of gastric juice.

The bowels are usually sluggish, and there may be obstinate pain. The quantity of urine is often considerably decreased, and this decrease is quite pronounced where there is frequent vomiting or in which considerable quantities of food are regularly removed from the stomach by aspiration. Quite frequently we see that the total quantity of urine passed in the twenty-four hours is as low as a liter or even half a liter. If the secretion of urine increases under treatment, this may be considered a good prognostic sign.

Other peculiarities of the urine are that it is frequently cloudy—that its specific gravity is increased; it often shows an alkaline reaction, and deposits a more or less abundant sediment of phosphates. All this need not surprise us. Sticker and Hübner[1] have shown by a series of exact experiments that there is a certain alternate relation between the secretion of gastric juice and the acidity of the urine. If no considerable production of gastric juice occurs after the entrance of food into the stomach, the reduction of the urinary acidity that should normally occur during digestion does not occur; for these reasons the normal fluctuations in the acidity of the urine are absent in inanition and in carcinoma, diseases in which the secretion of hydrochloric acid in the stomach is greatly reduced. On the other hand, abundant vomiting of large quantities of stomach-contents containing hydrochloric acid, or the continuous secretion of acid gastric juice, reduces the acidity of the urine. The same factors explain the reduction in the chlorids.

Vomiting, as we have mentioned above, is frequently seen in cases of continuous secretion of gastric juice. This symptom will be particularly severe and frequent if there is ectasy of the stomach at the same time; it may, however, occur without ectasy in the same way as in the intermittent form. Vomiting usually occurs at the height of the attack of pain. Many patients induce it artificially in order to gain relief; patients that are accustomed to the introduction of the sound frequently introduce the instrument themselves in order to remove the acid masses that are accumulating in the stomach. Vomiting at night, which we have mentioned above, is particularly characteristic and occurs in quite a number of cases.

In milder cases vomiting may be absent, even though there is continuous secretion of gastric juice, the pain gradually disappears, and no vomiting occurs. In these instances we must assume that the spasmodic contraction of the pylorus, which is usually caused by the secretion of much acid fluid into the stomach, gradually relaxes so that the fluid contents of the stomach can enter the intestine after a comparatively short time. If the stomach is pumped out at the height of the crisis, an abundant quantity of gastric juice will still be found. In a few cases of gastric crises in which vomiting did not occur diarrhea supervened.

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[1] *Zeitschr. f. klin. Med.*, vol. xii.

The quantity of the vomit varies : it may be very considerable in cases in which there is ectasy of high degree, and may amount to a liter or more, but even in those cases in which particles of food are not vomited, and in which there is no demonstrable retention of ingesta, the amount of vomited fluid may be very large. I have frequently observed the vomiting of 500 c.c. and more of pure gastric juice without any ingesta.

The vomit usually consists of a fluid mixed with a little mucus and frequently stained slightly green from the admixture of bile constituents. The patients state that it tastes acid or bitter. If particles of food are vomited,—that is, if vomiting occurs at a time when ingesta are still present in the stomach,—the vomit usually separates into three distinct layers : at the bottom of the vessel will be found a narrow layer of fine remnants of amylaceous material ; the chief portion of the mass—the middle layer—consists of a cloudy fluid ; on top will be found a third layer of foam of varying depth.

The reaction of the filtrate is always acid. It contains free hydrochloric acid and pepsin. The values for hydrochloric acid acidity may be either normal or increased ; the latter is seen particularly in cases where the vomit still contains morsels of food. The vomit usually possesses digestive powers and is capable of dissolving a disc of albumin in a normal manner. We refer to the section on the examination of aspirated stomach-contents for the special details.

Examination of the stomach is particularly important. We should not, of course, limit ourselves to the external examination, but should also test its secretion, its motility, and its absorptive powers.

In many, though not all, cases of continuous secretion of gastric juice the stomach is dilated. The longer the affection exists and the more severe the secretion of gastric juice, the greater, as a rule, the dilatation. Continuous secretion of gastric juice may occur without dilatation, as has been shown by Reichmann. I have myself repeatedly seen cases of this kind, and other authors, as Honigmann, Lichtheim, Wilkens, Strauss, and others, have reported the same. If the stomach is very much dilated, it is frequently dislocated at the same time—that is, it may either occupy a lower position or be more vertically placed than normal. It is an easy matter to demonstrate the presence of ectasy. If the stomach is inflated, the outlines of the organ can frequently be seen and felt ; at the same time succussion sounds may be elicited over an area that exceeds the boundaries of the normal stomach.

The fact that the stomach is distended does not necessarily indicate that ectasy exists, provided we define ectasy as an abnormal increase in the size of the stomach plus motor insufficiency. We can demonstrate that there is motor insufficiency of the stomach by examining the stomach-contents removed in the evening or early in the morning after fasting : in moderate degrees of motor insufficiency the stomach should always contain more or less abundant remnants of the midday meal if examined before supper; in more severe degrees some remnants of the supper will still be found in the morning before breakfast.

In other cases the stomach will be found to contain only gastric juice and no food-particles if examined in the morning after fasting and before breakfast. This does not, by any means, indicate that the motor power of the stomach is reduced. We are justified in speaking of a great reduction in the motor powers of the stomach only in those cases in which, in addition to gastric juice, an abundant quantity of food is found in the stomach after a prolonged period of fasting.

The significance of motor insufficiency and ectasy in cases of this kind is an important question to study. These complications may originate from two causes: they may either be due to simple ectasy of the stomach or they may be due to the presence of some mechanical obstruction at the exit from the stomach or in neighboring parts impeding the passage of the ingesta into the intestine. In the latter instance motor insufficiency is, of course, not absolute, but only relative.

I will refer later on to the connection between motor insufficiency and ectasy and continuous secretion of gastric juice, and will only mention in this place that, according to my own observations, both forms of ectasy can either follow the continuous secretion of gastric juice or occur at the same time with it. As far as I can judge from the history of cases of stenosis of the pylorus, the stenosis is not always the primary factor, but may occur secondarily as a result of other processes. A stenosis alone, as I shall presently show, is not sufficient to produce continuous secretion of gastric juice, even if hyperchlorhydria exists. That both conditions occur together does not demonstrate that the one is the result of the other.

Palpation shows that the region of the stomach is more or less sensitive to pressure; in some cases the stomach is sensitive to pressure over large areas; in other cases again, only in the region of the pylorus. Painful pressure-points are occasionally found in the back. No conclusions in regard to the seat of the trouble can be arrived at from the location of these pressure-points.

In many cases an increased resistance is felt or there is a circumscribed thickening in the region of the pylorus. I have already mentioned that cases of this kind, particularly if they are combined with severe degrees of ectasy, with great emaciation and loss of strength, render the diagnosis of carcinoma probable. The only way in which to avoid this wrong diagnosis is to examine the secretory functions of the stomach repeatedly. The cause of this circumscribed resistance is usually a cicatricial thickening of the region of the pylorus following an ulcer.

The examination of the stomach with the stomach-tube is more important than the external examination of the organ. The analysis of the stomach-contents must be modified to suit the questions that we are trying to determine in this section. The aspiration of stomach-contents for diagnostic purposes after a test-meal or a test-breakfast at the height of digestion is, of course, important; at the same time it is not sufficient in these cases, for here we wish, if possible, to demonstrate directly whether or not gastric juice is secreted in the absence of ingesta. For this reason the aspiration of stomach-contents must be performed at a time when the stomach is empty, or at a time when the mucous lining of the stomach has not been stimulated by food for a prolonged period.

Reichmann was the first to suggest examining the stomach-contents at a time when neither food nor drink had been introduced for a long period. He advised washing the stomach out thoroughly and withholding all food for from ten to twelve hours, and then determining whether or not appreciable quantities of gastric juice were present in the stomach. From what we have said it will be seen that this aspiration of stomach-contents for diagnostic purposes must be performed in different ways. I usually employ three procedures:

At first I aspirate the stomach-contents at the height of digestion. If ectasy exists, I aspirate the stomach-contents for from five to seven hours after a test-meal; I prefer aspirating after a test midday meal because a certain amount of information is given us by macroscopic examination at this time. If the absence of ectasy or of motor insufficiency can be determined, the aspiration of stomach-contents can be performed earlier. In the second place I wash out the stomach early in the morning after a period of fasting, and in the third place I wash out the stomach in the morning after fasting, and after the stomach has been thoroughly washed out the evening before at 10 o'clock, the patient, of course, taking neither solid nor liquid food in the mean time.

If a test-meal is administered to a patient with pronounced continuous secretion of gastric juice, and if the stomach-contents is aspirated for from four to seven hours later, an abundant, largely fluid, mass will, as a rule, be obtained. The quantity of this material varies, but may amount to one liter or more. If the stomach-contents is collected in a measuring cylinder, it will be seen that the mixture separates into three layers, like the vomit: on top there is a layer of foam; in the middle a cloudy, usually light-yellowish, fluid that forms the bulk of the mixture, and at the bottom a third layer, consisting of a fine whitish sediment. The latter consists almost exclusively of more or less finely distributed remnants of amylaceous material; morsels of meat are rarely found in the sediment; here and there possibly a few fine fibers, or, in exceptional cases, a few isolated coarse pieces of meat. Whether the presence of these larger morsels indicates insufficiency of pepsin secretion has not been determined; possibly in an ectatic stomach the mucous membrane arranges itself into folds in which some of these morsels become lodged and thus escape digestion.

The filtrate of the stomach-contents gives a very pronounced hydrochloric-acid reaction; the acidity is generally increased, and it may amount to 80, 100, and even more. The values for free hydrochloric acid equal 50 and more. The filtrate gives good peptone reactions and is capable of digesting a disc of albumin. Microscopic examination of the filtered residue reveals the presence of abundant masses of amylaceous material, many yeast-cells, and occasionally sarcina.

It is known that amylolysis begins in the mouth and continues for a time in the stomach, but that it is terminated as soon as free hydrochloric acid appears. In cases of continuous secretion of gastric juice different conditions exist in the stomach than in normal subjects, in the sense, namely, that free hydrochloric acid is present when the organ is empty ; besides, an additional amount of hydrochloric acid is produced as soon as food is taken, and this amount may be abnormally large because hyperchlorhydria is frequently found together with continuous secretion of gastric juice ; it is not surprising, therefore, to find that, under these conditions, amylolysis is impeded.

In many cases of continuous secretion of gastric juice we also see an abnormal development of gas, as shown by the layer of foam that forms on top of the stomach-contents. Kuhn has shown that this fermentative process may develop to such a degree that large quantities of combustible gas are formed. Gaseous fermentation always presupposes the existence of a considerable degree of motor insufficiency, and for this reason we find this symptom most pronounced in cases of continuous secretion of gastric juice complicated with severe degrees of motor insufficiency. This gaseous fermentation is due to the action of microbes, particularly of yeast-cells, on amylacea. A pronounced degree of fermentation indicates that stagnation of stomach-contents has occurred. The investigations of Kuhn, Strauss, and others demonstrate positively that the hydrochloric acid of the stomach-contents does not impede the development of yeast, provided a suitable pabulum for the development of these cells is given.

Strauss has reported an anomalous case of continuous hypersecretion that merits particular mention. In this case the vomit, as usual, formed three layers ; they were different, however, from those ordinarily seen : in place of a layer of foam, a layer of fat was found, consisting of numerous droplets of fat that were separated from one another by small swollen masses of amylaceous material ; a confluence of the different droplets of fat was prevented in this way. No gaseous fermentation developed in this stomach-contents even after the vomit was placed into the incubator. This case is remarkable because neither motor insufficiency nor ectasy could be determined, and because, nevertheless, quantities of stomach-contents that varied from 150 to 500 c.c. could be aspirated after a prolonged period of fasting, whether or not the stomach was washed out the evening before.

The examination of the stomach-contents after a period of fasting when the stomach is empty is more important than the examination of the stomach-contents at the height of digestion. The aspiration of gastric contents from an empty stomach can be performed in two ways : the stomach can either be washed out early in the morning before breakfast, or it can be first thoroughly cleansed the evening before and then washed out in the morning, care being taken, of course, that the patient eats nothing in the mean time. The former method is applicable only to those cases that are not complicated by severe degrees of ectasy. If ectasy is present, the stomach always contains morsels of food in the morning that have remained over from the day before. In cases of this character the first method cannot answer the question whether or not a secretion of gastric juice occurs when the stomach is empty. Here all food must be withheld for a long time or Reichmann's method be employed.

One objection that has been offered to this latter method, particularly in cases of ectasy, is that it cannot positively be determined whether or not the preliminary washing has really removed all food-particles from the stomach. The opponents of this method claim that even a small amount of food remaining behind would lead to the production of a certain amount of gastric juice, so that the presence of gastric juice in the morning would not necessarily indicate spontaneous secretion of gastric juice, but would merely be the expression of continuous digestive activity.

I believe, however, that it is possible to cleanse the stomach thoroughly, even in cases of most pronounced ectasy. I have performed this manipulation many times, and have arrived at the conclusion that it is practical. In many cases of ectasy, or, to employ the expression that is more popular with younger authors, in many cases of motor insufficiency of the second degree, I have thoroughly cleansed the stomach the evening before and then aspirated the stomach-contents the next morning before breakfast. Even in cases of most pronounced ectasy the stomach was often empty in the morning, whereas if the stomach was not cleansed the evening before, quantities of gastric contents varying from 500 to 800 c.c. were always found. I have records of a number of these cases. This, it appears to me, demonstrates that it is possible thoroughly to cleanse the stomach even if ectasy exists; it is true that to do this thoroughly much time and much patience are required; possibly, too, some practice and experience. No one will deny that remnants of food may remain behind if the lavage of the stomach is not carefully performed.

I think the statement that the presence of secretion in the stomach in the morning after evening lavage is an artefact may be considered a precarious one for another reason. Even assuming that, despite a most careful lavage in the evening, a few remnants of food could remain in the stomach, these particles could not be made responsible for the presence of an abundant amount of secretion if they disappear from the stomach by morning. It seems to me that as soon as these remnants of food would be propelled onward into the intestine, secretion in the stomach would stop. Secretion stops in a normal and even an ectatic stomach as soon as all remnants of food are removed from the organ. It might be argued that possibly the stomach had also succeeded in getting rid of the remnants of food that had remained behind from the evening before, but how would this explain that it did not, at the same time, get rid of the gastric juice that was secreted? It appears to me that if we find 150 to 200 or 300 c.c. or more of stomach-contents in the morning before breakfast without a trace of food, we are certainly justified in speaking of continuous secretion, even though ectasy be present. Simple ectasy, even when it is complicated with hyperchlorhydria and when it is highly developed, does not do this. It is true that the presence of a few particles of food may make the interpretation of the findings in the morning doubtful; but if 200, 300, or 500 c.c. of gastric juice should be found and only a few grains of amylaceous material, it appears to me that the amount of gastric secretion is not proportionate to the degree of irritation that these few grains of material could exercise on the mucous membrane of the stomach. A characteristic feature of continuous secretion of gastric juice is the disproportion between the irritation and the secretion. If swallowing saliva causes the appearance, in a perfectly healthy person, of 300 c.c. of gastric secretion in the morning, we must assume that there is an abnormal irritability of the gastric glands. The same obtains in cases in which an acute gastrosuccorrhea occurs after some psychic excitement: here the gastric glands are overstimulated by an irritant that ordinarily, and under normal conditions, would not exercise any effect on their function.

But I will leave aside those cases in which a few isolated particles of food are found in the morning, together with an abundant quantity of gastric juice. We may, at all events, say that a large quantity—100 c.c. and more—of gastric juice without any admixture of food-particles indicates continuous secretion of gastric juice, particularly if found in the morning after thorough lavage of the stomach the evening before. If we know that the wash-water of the evening before was perfectly clear and did not contain a trace of hydrochloric acid, and that the patient ate nothing whatever during the night, we are certainly fully justified in diagnosing this condition. It is immaterial whether this state is called spontaneous or continuous secretion of gastric juice; at all events, the stomach in these cases reacts to stimulants that ordinarily would not irritate it, and an abundant secretion of gastric juice occurs where otherwise only a minimal quantity or none at all would be secreted.

After all, I consider Reichmann's method, provided it is performed with all necessary precautions, practical; at all events, I always use it in cases of this kind. It is necessary, of course, that the fluid aspirated, the so-called "fasting secretion" ("Nüchternes Sekret"), possess all the properties of gastric juice. The term *secretion* after fasting is used in contradistinction to the term *residue* after fasting; the fluid aspirated is usually watery and clear or slightly yellowish-green in color, and frequently turns grass-green on standing. The green color is usually due to the admixture of small quantities of bile constituents. The secretion generally consists of a slightly cloudy fluid, and contains no threads of mucus unless some complication exists. The reactions for free hydrochloric acid are usually distinct. The values for hydrochloric acid, also those for free hydrochloric acid, are, as a rule, pretty high; no organic acids are found. The biuret reaction is positive; no gaseous fermentation occurs even in the incubator after the addition of dextrose; the secretion possesses peptic powers. According to the investigation of Strauss, performed in my clinic, the specific gravity may be from 1004 to 1006.5, or only a little higher; the residue after fasting, on the other hand, has a much higher specific gravity.

In those cases in which pronounced degrees of motor insufficiency do not exist things will be different; here aspiration in the morning before breakfast reveals the presence of a homogeneous secretion, provided the stomach has the power of removing the ingesta during the night. The morning secretion will be the same, whether the stomach was carefully washed out the evening before or whether lavage was not performed after supper. The following case may be quoted as an example: The man, L. F., was an agriculturalist forty-nine years old. When the patient was first received in the hospital, the clinical diagnosis of gastroptosis and hyperacidity was made; he was examined later on and a second diagnosis of gastroptosis, slight motor insufficiency, and continuous secretion of gastric juice was substituted.

The patient was received in the hospital in August, 1893. The first history elicited was altogether negative, the patient stating that he had always been perfectly healthy. The first stomach symptoms appeared after the campaign of 1866; the symptoms receded from time to time, but would always recur. The only distressing symptoms were attacks of pain; only in exceptional cases, "if there was too much acid," vomiting occurred; as soon as he ate something the pain disappeared. Objective examination revealed that the stomach was slightly lower than normal, with its lower boundary about a finger's width below the umbilicus. The quantity of residue that could be aspirated after a test-meal was small: in one case 75, in another 120 c.c. The acidity was increased—the total acidity 120, free hydrochloric acid 73 to 79. In the morning, after fasting, the stomach was empty, also if it was thoroughly cleansed the evening before. The presence of gaseous fermentation could not be determined.

Aspiration in the morning after fasting and before breakfast revealed the following conditions:

	Gastric secretion.	Specific gravity.	Total acidity.	Free hydrochloric acid.
Nov. 26. Without cleansing the stomach the evening before, after an abundant supper	250 cm.	1005.0	80	53
Nov. 27. After washing out the stomach the evening before	110 cm.	1004·5	67	47
Nov. 28. Without washing out the stomach the evening before	200 cm.	1005.0	58	36
Nov. 29. Without washing out the stomach the evening before	200 cm.	1005.0	68	52
Nov. 30. Without washing out the stomach the evening before	200 cm.	1004.5	75	50

In this case, therefore, 110 to 250 c.c. of secretion were found on the following morning, whether or not the stomach was cleansed the evening before, or whether an abundant supper was given, followed by cleansing of the stomach. The stomach-contents contained no particles of food, and its specific gravity fluctuated between 1004 and 1005.

During his first visit to the hospital the patient remained only five days. About a year afterward he was again admitted; he stated that he had felt very well after leaving the hospital, but that his old symptoms had returned. He suffered no pain for two or three hours after a large meal; at the end of this time, however, the pain would begin, and reach its acme about six hours after eating. He also complained of pain between 11 and 2 o'clock at night, also occasionally before eating. In the course of the last four weeks attacks of vomiting occurred almost regularly—in the night between 11 and 2 o'clock, and in the evening between 5 and 6 o'clock. He claims that his appetite, which formerly was so good, is now very much reduced. Occasionally he develops canine hunger. An objective examination reveals that the stomach is lower than normal—the upper boundary 2 fingers above, the lower one 3 to 4 fingers below, the umbilicus.

Aspiration of the stomach-contents after a test-meal showed that from 300 to 400 c.c. of residue were present containing remnants of amylaceous material, no meat, no yeast-cells; there was no fermentation. The total acidity was 150; free hydrochloric acid, 40.

Here, then, a continuous secretion of gastric juice had developed. When the patient first entered the hospital this condition could not be diagnosed; at that time, however, he was already suffering from hyperchlorhydria. When he was admitted the second time, there was a moderate degree of motor insufficiency; the insufficiency, however, was not severe, as was shown by the absence of food-particles in the morning; nevertheless, a fairly abundant quantity of secretion was always present in the morning after fasting and before breakfast. That the secretion was not due to the lavage of the stomach was shown by the fact that an analogous amount of secretion containing no particles of food was found in the morning, even if the stomach was not washed out the evening before.

Inversely, I could report a number of cases of motor insufficiency of high degree plus hyperchlorhydria in which a large amount of residue was found in the morning when the stomach was not thoroughly washed out the evening before, whereas nothing was found if the stomach had been washed out. It may suffice to mention one example:

Mrs. A. W., aged thirty-seven, underwent a laparotomy for an ovarian cyst four years ago. Some time afterward she complained of pain in the right side that appeared particularly when she walked. Soon attacks of vomiting occurred three and four hours after eating, which grew more and more frequent; pain appeared in the epigastric region, and she suffered from heartburn. These symptoms were relieved by vomiting; occasionally the disagreeable symptoms would disappear if the patient took some more food. Hematemesis never occurred. There was no nocturnal pain. The bowels were constipated, the thirst increased.

Physical examination revealed sensitiveness to pressure in the right hypochondriac region radiating toward the scar. There was a very distinct succussion sound in the left hypochondriac region and in the epigastrium, extending three fingers below the umbilicus. Abnormal resistance could not be felt anywhere.

The stomach-contents was aspirated one hour after a test-breakfast; 210 c.c. of finely divided stomach-contents were found. The total acidity was 81; free hydrochloric acid, 90. Four hours after a test-meal 400 c.c. of a finely distributed mass of food and an abundant quantity of fluid were found; total acidity, 120; free hydrochloric acid, 85. Inflation showed that the lesser curvature was situated three fingers above the umbilicus, the greater curvature two fingers above the symphysis.

Aspiration of the stomach-contents early in the morning, preceded by lavage of the stomach the evening before (the patient taking the last food at 9 o'clock in the evening), showed that 600 c.c. of stomach-contents were present that readily separated into three layers. Microscopic examination revealed the presence of numerous grains of starchy material, sarcina, and yeast-cells. The total acidity was 160; free hydrochloric acid, 76. In the incubator there was an abundant development of gas. The next evening the stomach was thoroughly washed out at 10 o'clock. Aspiration the next morning showed that the stomach was completely empty. Methodic lavage was instituted, massage was given, a suitable diet prescribed, and other measures employed. Under this régime the residue in the evening became smaller and the motor insufficiency improved. About seven weeks after her admission into the hospital the stomach was found completely empty in the evening before supper. After ten weeks she was dismissed, cured of all her symptoms; the tone of the organ was normal; the stomach was still slightly dislocated downward.

The clinical diagnosis of this case when it was admitted was ectasy, motor insufficiency of the second degree, gastroptosis, hyperchlorhydria, no continuous secretion of gastric juice.

In this case, therefore, ectasy of high degree and hyperchlorhydria had been present for some time; notwithstanding this no continuous secretion of gastric juice was found. Even though the stomach was carefully washed out the evening before and though the conditions were most favorable for stagnation, and although, finally, hyperchlorhydria existed, no gastric juice was found in the morning. This demonstrates, like the other case quoted above, that it is possible thoroughly to cleanse an ectatic stomach, and that ectasy alone, even when it is combined with hyperchlorhydria, is not sufficient to cause the secretion of gastric juice; a second factor must, therefore, always be present in addition to ectasy: wherever we find continuous secretion of gastric juice in this condition, we must assume that the glands of the gastric mucosa are abnormally irritable.

It is possible to demonstrate in still another way that the continuous secretion of gastric juice is not, as has been stated, due to stagnation and to the prolongation of digestion and the secretion of gastric juice that naturally would result from such a condition.

We have repeatedly introduced the stomach-tube in cases with continuous secretion of gastric juice without, at the same time, introducing anything from the mouth into the stomach, and were still able to aspirate some secretion from the stomach every time the sound was introduced, even though the intervals between the introduction of the sound were very short. I will limit myself to quoting one example of this kind:

	Number of cubic centimeters.	Specific gravity.	Total acidity.	Free hydrochloric acid.
I. Contents of the stomach in the morning after fasting and before breakfast and after the stomach was thoroughly cleansed the evening before	200	1005.0	42	32
Half an hour after the first aspiration	200	1004.0	72	55
One hour after the first aspiration	290	1004.0	71	62
An hour and a half after the first aspiration	190	1004.0	50	40
II. Contents of the stomach in the morning before breakfast, after fasting, and after lavage the evening before	150	1004.5	80	51
Half an hour later	100	1005.0	75	58
One hour later	100	1005.0	66	44

I wish expressly to emphasize the fact that the same amount of fluid was found in the stomach the next morning after fasting and before breakfast, whether or not the stomach was washed out the evening before. The constitution of the fluid, moreover, was the same, and in neither case did we find any particles of food. The fact that a considerable quantity of gastric secretion could be aspirated after so short a time shows conclusively that in this case the accumulation of secretion was not due to a stagnation of the product of the gastric glands, but that a new quantity of gastric juice was continuously secreted ; the fact, also, that only a few hundred cubic centimeters of gastric juice were found in the morning after fasting and before breakfast demonstrates that in the mean time some secretion must have been removed from the stomach ; in other words, that this finding cannot possibly be attributed to simple stagnation. It seems justifiable to inquire why this secretion was not at once completely removed from the stomach. We must expect, however, *a priori*, that if the secretion of gastric juice is continuous, a portion of the secretion will remain behind, particularly in cases of ectasy. The examples we have quoted above show at the same time that ectasy alone, even if it is very severe, cannot cause the continuous secretion of gastric juice, even if hyperchlorhydria exists at the same time.

It is not surprising to find small quantities of combined hydrochloric acid in these conditions ; even in normal subjects, in whom a certain amount of gastric secretion is occasionally found after fasting, hydrochloric acid is occasionally in combination. It is possible that the saliva, mucus, etc., that has been swallowed can be made responsible for this. The saliva that is swallowed cannot, however, be considered the cause of continuous gastric secretion ; this is manifest when we remember that normally appreciable quantities of gastric juice are never found after fasting. We are justified in deducing, therefore, that another factor—namely, abnormal irritability of the gastric glands—must play a rôle.

After all that we have said I feel justified in stating that the presence of an abundant quantity of gastric juice after a period of fasting proves that there is a continuous secretion of gastric juice. I also feel justified in indorsing Reichmann's method of washing the stomach late at night, then pumping it out early in the morning before breakfast. I have found this method very useful and can recommend it. The aspiration of stomach-contents must, of course, be carefully and thoroughly performed ; but in case a little water should remain behind, we may expect that it will be propelled into the intestine before the next morning. In ectasy of severe degree, even if it is combined with hyperchlorhydria, the stomach is usually found empty in the morning if it is thoroughly washed out the evening before, but assuming that some water were retained, this alone would never cause a continuous secretion of gastric juice even in ectasy ; if it did, we would be justified in believing that so mild an irritant as luke-warm water was capable of causing a permanent production of gastric juice.

I will admit that in certain cases of severe ectasy or in cases of motor insufficiency of high degree it may at times be impossible to remove all the water from the stomach; but if in the one group of cases we constantly find some secretion in the stomach the next morning, and do not find it in the other group of cases in spite of the fact that the same manipulations are performed in both cases, and that in both the degree of motor insufficiency is the same and the secretion of gastric juice is good; in fact, even though there be hyperchlorhydria, we must conclude that something besides ectasy is responsible for this continuous secretion.

I have already mentioned that in cases of continuous secretion of gastric juice nocturnal attacks of pain are quite frequent. This seems to me to be particularly significant in those cases in which the motor powers of the stomach are not reduced or are only slightly impaired. Here the occurrence of such nocturnal attacks leads us to suspect some secretory perversion, for pain can never be produced in the stomach when it is empty. If, for instance, attacks of pain with vomiting occur in a case whose motor power is good or only slightly reduced, and at a time when we can assume that the food eaten at supper-time is removed, we may readily suspect that there is spontaneous secretion of gastric juice; attacks of pain, therefore, at 2 o'clock in the morning after a simple supper indicate the existence of this perversion. If the patient is afflicted with motor insufficiency of high degree, this conclusion is not, of course, valid.

If what we have said is correct, namely, that the continuous secretion of gastric juice is no disease *sui generis*, but merely a perversion of function, we need not be surprised to find numerous other complications in this condition.

Complications.—The most frequent complication we encounter is ectasy; this may be either purely atonic or may be due to stenosis of the pylorus. The fact that continuous secretion of gastric juice is frequently seen without ectasy indicates that the latter condition is not the direct cause of the former; this is further demonstrated by the fact that continuous secretion of gastric juice is absent in the majority of cases of ectasy. If the development of the disease can be carefully pursued, it will frequently be found that ectasy does not complicate the process until late in the disease. If the patient is seen after the disease-picture is fully developed, it is almost impossible to determine whether the ectasy or the perversion of gastric secretion was the primary event.

The development of atonic ectasy in cases of continuous secretion of gastric juice can readily be understood; the fact that gastric juice is continuously present in the stomach and that this fluid is very acid and that its acidity is increased whenever more food is introduced, must naturally impede amylolysis; consequently amylaceous food remains in the stomach for an abnormally long time. Another reason why the stomach never empties itself is that the secretion of gastric juice is so continuous that it can never be completely evacuated and the stomach can never be absolutely at rest. A third factor that favors the development of ectasy is the increased acidity of the stomach-contents, which either at the height of digestion or possibly when the stomach is empty may cause spasmodic contraction of the pylorus; when this occurs, the propulsion of the stomach-contents into the intestine is prevented. Owing to this continuous strain on the organ, which need not be very severe in the beginning, the stomach gradually becomes exhausted, its walls relaxed, and gradually more and more distended, so that in this way ectasy develops.

The explanation of this process is very difficult in those cases in which there is a stenosis of the pylorus and in which the peristaltic movements of the stomach are very active, showing that the stomach, while dilated, is not atonic, but hypertonic. It is impossible to decide in this case whether the stenosis of the pylorus was the primary factor or whether it was secondary to the continuous secretion of gastric juice, unless, of course, the development of the disease has been carefully studied from its incipiency. To judge from my personal experience, both classes of cases can occur, but even in those instances where the stenosis of the pylorus is the primary factor and the continuous secretion of gastric juice the secondary one, I do not think it by any means positively demonstrated that the latter condition is a direct result of the former. There may be stenosis of the pylorus of very high degree, even stenosis of the pylorus plus hyperchlorhydria, and at the same time no continuous secretion of gastric juice; again, there may be much stagnation of stomach-contents, the stomach may be so ectatic that it cannot get rid of its contents over-night, and still there may not be any continuous secretion of gastric juice whatsoever. We may say, therefore, that neither hyperchlorhydria nor ectasy of high degree is necessarily followed by a continuous secretion of gastric juice.

It is true, as we have shown above, that hyperchlorhydria and continuous secretion of gastric juice, while different in many respects, are related in others; and if it is true that hyperchlorhydria can result from a slight degree of hyperirritability of the gastric glands, and continuous secretion of gastric juice from a high degree of irritability of these glands, we need not be surprised, in some instances, to see a case of hyperchlorhydria gradually develop into a case of continuous secretion, and in other instances to see no such development. Hyperchlorhydria is the most favorable predisposing factor of ulcer. It is pretty generally recognized nowadays that ulceration of the stomach readily develops in cases of hyperacidity. If a patient is afflicted with continuous secretion of gastric juice, there is usually some hyperchlorhydria; we need not be astonished, therefore, to find that a patient with continuous secretion of gastric juice readily develops ulcer; if such an ulcer is situated near the pylorus, a cicatricial stenosis of this part may develop. This may explain why an ulcer may secondarily develop in cases of continuous secretion of gastric juice, leading in its ultimate consequences to a cicatricial stenosis of the region of the pylorus and to secondary ectasy.

Things are different in cases in which ectasy is apparently the primary factor, gastrosuccorrhea the secondary one; here, it appears to me, we are justified in assuming that the formation of an ulcer is favored by the hyperacidity, that the ulcer leads to stenosis of the pylorus and then to ectasy. Ectasy *per se* did not cause the continuous secretion of gastric juice; it is more probable that the hyperchlorhydria that existed became exacerbated until it developed into continuous secretion of gastric juice; this exacerbation, we can imagine, was favored by the stagnation of stomach-contents. At the same time we have shown that this sequel of events need not necessarily occur.

If there is much stagnation of stomach-contents in a case of ectasy, continuous secretion of gastric juice may undoubtedly be simulated. The only way to determine whether we are really dealing with continuous secretion of gastric juice or not is to analyze the stomach-contents after a period of fasting and when the stomach contains no food-particles.

Some authors have attempted to prove that the continuous secretion of gastric juice is merely the result of stagnation of ingesta; they quote cases that were cured of continuous secretion of gastric juice by methodic lavage of the stomach and by restricting the administration of food *per os*. This argument is not valid, however, for treatment of this kind necessarily spares the stomach, so that not only the motor powers, but also the secretory powers, of the organ are improved. To judge from my personal experience, the secretion of gastric juice does not stop if food is withheld; in my cases the secretion continued for quite a time, so that I cannot agree with those authors who claim that it is arrested.

The explanation we have offered above furnishes the most simple explanation for all cases; possibly arguments may be adduced against it, but I cannot imagine a more plausible one. I feel justified in concluding, from my personal experience, that the development of this disease is not the same in all cases.

A second complication, which I have already mentioned, is ulcer. Directly connected with this lesion are the gastric hemorrhages that are occasionally observed; we are not justified, however, in attributing any hemorrhage from the stomach that is observed in a case of continuous secretion of gastric juice to the existence of an ulcer. We must always remember that the mucous membrane of the stomach is in a condition of excessive irritation, so that hemorrhage may readily occur even without ulceration.

Some clinicians have attempted to prove that in continuous secretion of gastric juice the mucous lining of the stomach is particularly irritated; they refer to the fact that in these cases small particles of mucous membrane are frequently torn off when the stomach-tube is withdrawn. I am willing to admit that in cases of this kind the mucous membrane of the stomach is more congested than normal, more swollen, and somewhat loosened; I believe, however, that such an accident as tearing off particles of mucous membrane when the sound is withdrawn can usually, probably always, be avoided. I see almost daily how physicians make the fundamental mistake of withdrawing the sound, after performing lavage, as soon as the last drop of water has run into the stomach; instead of letting the sound go, they compress it tightly and then withdraw it; under these circumstances a piece of mucous membrane may very readily be drawn into the openings of the sound and torn off. The same, however, may occur in a normal stomach if the same technical error is committed. If care is taken that the sound is not compressed when it is withdrawn, the mucous membrane will not be torn; in fact, I think it is hardly possible to aspirate the mucosa into the openings of the sound and to lacerate it if the sound is not compressed.

Another complication that is occasionally, though rarely, seen in continuous secretion of gastric juice is tetany.

Kussmaul, in 1869, was the first to call attention to the occurrence of tetany in dilatation of the stomach. Within recent years a large number of analogous observations have been published; the majority of these instances refer to cases of continuous secretion of gastric juice. Tetany, however, is also seen in other forms of dilatation of the stomach. The statement of Bouveret and Devic that tetany occurs only in cases of dilatation of the stomach that are complicated by continuous secretion of gastric juice is not quite correct. In regard to this point I refer to page 163, where I have reported 2 cases of tetany I observed myself; both occurred in cases that were in early stages of carcinoma of the pylorus. Blazicek has reported a case in which the ectasy of the stomach was due to compression of the duodenum by the dilated gall-bladder. From all this we learn that tetany is not a form of spasm that occurs only in continuous secretion of gastric juice; it is true, at the same time, that in the majority of cases of tetany this condition of the stomach will be found; in all cases there was at the same time a severe degree of ectasy.

That this complication is not a frequent one is shown by the small number of cases that have so far been reported; all in all, there are hardly 30. The personal experience of a number of clinicians also shows that the condition is rare; personally, I have seen well-developed tetany only in 3 cases of dilatation of the stomach. This is a remarkably small proportion when I consider the large number of cases of ectasy that I have had under observation; in nearly all my cases there was ectasy of high degree and the patients were very much emaciated.

I presuppose a knowledge of the symptoms of tetany. In all the cases the well-known attacks of tonic spasms of the extremities were seen, recurring in long or short intervals, and in certain cases extending to the muscles of the face, the neck, and the body. Other characteristic symptoms of tetany are Trousseau's symptom, the facialis phenomenon, the increase in mechanical irritability, etc.

I refer to the general part of this work for a discussion of the theories that have been pronounced on the origin of tetany. Tetany constitutes one of the most serious complications, as nearly two-thirds of the cases reported terminated fatally.

Course.—The course of a case of continuous secretion of gastric juice is by no means uniform. Mild cases can be distinguished in which the symptoms can all be explained by the perversion of secretion alone, and more severe ones in which there are ectasy of high degree, ulcer, and similar complications. The course of the disease will naturally vary according to the presence or absence of these complications. The majority of clinicians speak only of the last-named group; the former class of cases is, however, not rare by any means; it is true they can easily be overlooked if the stomach-contents of these patients is not examined after a period of fasting. Mild cases of the former type, provided they have existed only for a few weeks, can often be cured in a short time; it is another question whether the cure of these cases is permanent.

In a more severe type of cases motor insufficiency frequently develops after a time, and this may ultimately lead to severe degrees of ectasy. In cases of this character the symptoms are correspondingly severe, the pain recurs regularly in the evening and during the night, vomiting occurs almost daily, the patients are frequently very much emaciated, exceedingly weak, and languid. If the disease is complicated by repeated hemorrhages from the stomach, or if stenosis of the pylorus, adhesions, or other lesions develop as the result of ulceration, the course is naturally still more unfavorable. If there is much stagnation of stomach-contents, this may constitute a new source of irritation and lead to a persistence of the continuous secretion of gastric juice.

Very severe cases may terminate fatally from exhaustion; or a severe hemorrhage from the stomach may occur and endanger the life of the patient. In rare cases tetany closes the scene. On the other hand, cases afflicted in this way may live for many years. The course of the disease will naturally depend on the treatment and on the general mode of life of the patient.

Prognosis.—The prognosis is always doubtful. Mild cases in which there is no complication frequently leave the hospital cured; in many instances, however, they return for treatment with all the old symptoms. I can state positively that some of my patients were free from all distress two years after they were dismissed; whether they were permanently cured is another question.

The majority of cases that enter hospitals are old and complicated cases; here the prognosis is never altogether favorable, but even these chronic cases may be improved. I remember a case in which I was able to diagnose continuous secretion of gastric juice, stenosis of the pylorus, and ectasy of high degree some twelve years ago. There was a circumscribed area of thickening that could be felt in the region of the pylorus. According to the history that this patient gave, the symptoms began six years before I saw him. A short time before the patient consulted me the diagnosis of carcinomatous stenosis of the pylorus had been made by another physician. As a matter of fact, the patient was so much emaciated and so cachectic when I saw him that this diagnosis seemed justified, particularly as repeated attacks of hematemesis had occurred a short time before; other attacks of violent bleeding from the stomach then occurred, so that there was little hope of restoring the patient to health. To-day this case is still afflicted with ectasy of high degree, there is pronounced hypersecretion, and he is obliged to wash out his stomach several times a day and to limit himself to a carefully selected diet; at the same time he is fully able to attend to his work. No one would suspect that this man is afflicted with so serious a trouble, and although he is sixty years of age, he is capable of taking long walks for hours without overexerting himself. The only way, of course, in which so favorable a result could be brought about was to place the patient on a very carefully selected diet, and I fear that many persons in his position would not have carried out the dietary regulations so readily or would not have been able to comply with the rules laid down, owing to external circumstances. I might add that in this case the advisability of operative interference, which we will discuss in the section on Therapy, was repeatedly considered, and that it was not undertaken simply because the patient obstinately refused to allow any surgical procedure. We will discuss below what might have been expected in this case from surgical measures.

Diagnosis.—There is comparatively little to say in regard to the diagnosis. In this disease the diagnosis is easy if the typical symptoms are looked for; unfortunately, they are frequently overlooked. If a patient presents himself with a disease-picture similar to the one delineated by Reichmann, the disease under discussion may be suspected, but that is not enough. Attacks of pain occurring both at the height of digestion and in the night, increased thirst, canine hunger, good meat digestion, deficient amylaceous digestion, vomiting of abundant quantities of fluid, the separation of the vomit and of aspirated stomach-contents into three layers, may all cause the physician to suspect the existence of continuous secretion of gastric juice. At the same time, this diagnosis can be positively made only if it can be directly determined that an abundant secretion of gastric juice occurs even when the stomach contains no food whatever. We have already explained at length how this proof can be furnished, and will not refer to it again in this place.

If the existence of continuous secretion of gastric juice has once been determined, it remains to be seen whether it is transitory, intermittent, or permanent. A probable diagnosis can usually be made from the symptoms, for in the one case the general symptom-complex appears at irregular intervals; in other cases it appears regularly, but with varying intensity. This question, too, can be decided positively only by repeated aspiration of the stomach-contents when the stomach contains no food or after a prolonged period of fasting, after the stomach has been thoroughly cleansed some time before. This affection can be confounded with others only if these methods of examination are omitted.

I have already mentioned that there are certain cases in which the totality of symptoms is so similar to that of carcinoma that an experienced observer may make an erroneous diagnosis. This, of course, cannot occur in cases of simple uncomplicated continuous secretion of gastric juice, but only in those cases that are complicated with ulcer or cicatricial stenosis of the pylorus following ulcer and ectasy. But even in these cases it is frequently possible to make a differential diagnosis without examining the stomach-contents, and certain distinguishing symptoms can usually be discovered that do not fit into the frame of carcinoma. None of these symptoms, however, is altogether decisive; I refer, for instance, to the absence of aversion for meat. An experienced observer will doubt the diagnosis of carcinoma if, on examining the stomach-contents, he finds that it separates into three layers, that it contains no remnants of meat, but abundant quantities of amylaceous material, and if he finds that the vomit presents essentially the same appearance. All this negative evidence will, of course, induce him to perform careful examinations of the gastric secretion.

It is almost impossible to confound this condition with ulcer. On the other hand, a picture very similar to it can be produced by cicatricial stenosis of the pylorus with secondary ectasy of the stomach; some authors, in fact, have gone so far as to state that continuous secretion of gastric juice is nothing else than the ordinary ectasy of older authors. It is hardly possible to determine whether or not the older authors only call those cases ectasy in which there was continuous secretion of gastric juice, for in their time the secretion of gastric juice was never examined; ectasy *per se*, however, is only a symptom, a sequel, and not a disease in itself. If for no other reason, this should debar anybody from claiming that continuous secretion of gastric juice corresponds to the picture of ectasy. A case of cicatricial stenosis of the pylorus with ectasy certainly develops many symptoms that are also seen in chronic secretion of gastric juice. This is natural, for we see continuous secretion of gastric juice together with an ulcerative cicatrix or ectasy; but even though ectasy and stenosis of the pylorus exist, the symptoms are not altogether identical. Above all, those symptoms are absent that are most indicative of the existence of continuous secretion of gastric juice. It is absolutely necessary to perform the decisive tests, for otherwise it is very easy to confound the different conditions, particularly as the stomach is never empty in severe degrees of stenosis of the pylorus, and the continuous presence of hydrochloric acid in the stomach-contents must naturally impede the digestion of amylaceous material as much as in cases of continuous secretion of gastric juice. Despite all objections that have been formulated against my view, I still maintain that if physicians will proceed as I have indicated, the results obtained from their examinations will be positive. It is not well, however, to rely altogether on the results of one examination; the analyses should be repeated, and, if necessary, modified to suit the individual requirements of the case.

Pathologic Anatomy.—I have already mentioned, in the introduction, that the symptom-complex that we group under the name of continuous secretion represents merely a perversion of function. In certain cases of this disease we naturally may expect to find anatomic changes;

it is questionable, however, whether the anatomic findings will be uniform. An increase of glandular secretion may be produced in different ways: it may be due to nervous influences,—for instance, to direct or reflex irritation of the nerves of secretion,—or it may be due to inflammation of the mucous lining of the stomach, or to many other conditions.

Korczynski and Jaworski[1] deserve the credit of having performed some very careful investigations into the anatomy of the gastric mucous membrane that they removed during operations on cases of gastric ulcer that were complicated with continuous secretion of gastric juice. Macroscopically, they found a thickening of the mucosa, with more or less pronounced "*etat mamelonné*"; microscopically, they saw small-cell infiltration underneath the raised surface epithelium, which extended downward between the interglandular tissues into the submucosa and even penetrated the tubules of the glands; the peptic cells were degenerated and disintegrated, whereas the parietal cells were well preserved.

In those cases in which the last-named changes were especially pronounced, the outline of the different glandular tubules was indistinct, and in their place areas of small-cell infiltration were seen, within which were found numerous well-preserved parietal cells.

Korczynski and Jaworski state that gastrosuccorrhea may be present without any anatomic lesions of the stomach mucosa. Inversely, we may expect that if irritation of the mucous lining of the stomach continues for a long time, particularly if there is stagnation of ingesta at the same time, certain gastric changes may develop; there may be small-cell infiltration underneath the surface epithelium that may extend further down and ultimately may reach the submucosa; at the same time there will be hyperemia, so that the peptic and parietal cells will be overactive, so that there will be an increased secretion of pepsin and of hydrochloric acid. The presence of hydrochloric acid in an empty stomach exercises a deleterious effect on the glandular tubules, and as a result of all these noxious agencies the peptic cells perish.

This anatomic picture corresponds to the clinical findings, for Jaworski and Korczynski were able to determine in their cases that numerous cylindric epithelia were present in the empty stomach in addition to disintegrated cell-nuclei, mucous cells, etc. Hayem has described changes similar to those found by the above-named authors.

It is to be expected *a priori* that in the course of disturbances of this kind such changes in the mucous lining will occur; it is another matter to decide which is the primary, which the secondary, event. For the present it appears to me that we are not justified in regarding continuous secretion of gastric juice as a form of chronic gastritis. As arguing against such a view, we might mention the fact that the symptoms occasionally appear suddenly, that they frequently intermit, and that the composition of the secretion is not what we should expect in gastritis. In hyperchlorhydria we also find changes in the mucosa of the stomach under certain conditions; this does not, by any means, demonstrate that hyperchlorhydria is always a result of these changes, for we know that hyperchlorhydria is frequently a purely functional disorder. The same probably applies to the continuous secretion of gastric juice, although we cannot deny that quite frequently, particularly in advanced cases, certain anatomic changes will be found.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xlvii.

Therapy.—The first object of treatment is to apply remedies that will suppress the continuous secretion of gastric juice. The most rational procedure would be, at least in cases of very severe degree, to remove all irritation from the gastric mucosa for a prolonged period of time—in other words to stop all feeding by mouth and to nourish the patient exclusively by the rectum.

We know of no remedy that will positively suppress the continuous secretion of gastric juice. Reichmann has recommended lavage of the stomach with solutions of silver nitrate in the strength of 1 to 2 : 1000, and claims that this treatment will limit the excessive secretion of gastric juice. Other authors have recommended the internal administration of silver nitrate. Personally I have never seen any good results follow this treatment. Atropin and morphin have been recommended for suppressing the secretion; similar virtues have been attributed to Carlsbad waters and alkaline remedies in general, and it has been claimed that they directly influence the secretion of gastric juice. It cannot be denied that they possess certain powers, but the good effects they exercise are not due to any influence on the secretion of gastric juice, but merely to the neutralizing action they have on the gastric juice, or, better, the acid of the gastric juice, after it is secreted.

Where the cause of the perversion can be definitely determined, and where it is possible to remove this cause, such a course of treatment should be attempted in the first place. It will rarely, however, be possible to determine the primary and original cause of the perversion. All the causes that we have enumerated in the section on the etiology of continuous secretion of gastric juice, as insufficient mastication of food, rapid eating, and other similar abuses, are merely predisposing factors. It is altogether undetermined why these predisposing factors cause gastritis in one case, atony in another, and continuous secretion of gastric juice in a third person. At all events, however, every effort should be made to cure the patient of any bad habits of this character, and, if possible, to remove any one of the predisposing factors we have mentioned.

The most important rule of treatment is to administer food that will not stimulate the secretion of hydrochloric acid any more than necessary, but that will at the same time contain sufficient nourishment; a diet, moreover, should be administered that will not overload the stomach. All strongly irritating substances, as mustard, paprica, pepper, radishes, acids, strong alcoholics, etc., must naturally be avoided; extreme temperatures, very hot or very cold articles of food and drink, should be directly interdicted.

Since these patients, as we have shown, digest amylacea badly and albuminous food comparatively well, the composition of the diet should be arranged accordingly; the patients should be given chiefly albuminoid and gelatinous substances, and the amylacea should be reduced as much as possible. In many respects these dietary regulations correspond with those that are employed in diabetes. If starchy food is given at all, it should be given only at a time and under conditions when it can be well digested; if it is introduced into the stomach in large quantities when the organ is full, it is not utilized at all, or only to a very slight extent. If the stomach is previously washed out so that no acid is present, some of the starchy food can be digested. It is wrong, therefore, to say categorically that patients with hypersecretion should eat no starchy food; it is more conservative carefully to explain to them when and under what conditions they may eat it.

Another factor that must be considered in selecting the diet is whether or not ectasy is present; the dietary regulations will also differ somewhat according to the origin of this ectasy, and will be different when the ectasy is due to stenosis of the pylorus and when it is a simple atonic form.

The general strength of the patient must also be considered in treating these cases. In a patient with moderate hypersecretion whose strength is good the treatment will be different from that used with a patient who is extremely emaciated and reduced.

The following regulations apply to a case of moderate hypersecretion without ectasy or with a slight degree of ectasy. In the morning one to two teaspoonfuls of Carlsbad salt in a cup of warm water are taken before breakfast; as there is always a certain amount of secretion in the stomach in cases of hypersecretion, part of the acidity is neutralized, and, besides, the stomach-contents is propelled into the intestine; at the same time constipation, if it is present, is relieved. The salt, therefore, acts approximately like lavage of the stomach. If desirable, the stomach may, of course, also be washed out. I am in the habit of performing lavage only in those cases in which there is ectasy of high degree and in which particles of food are found in the stomach early in the morning. If ectasy is absent or only moderate, I prefer administering Carlsbad salts; an hour later the patient may sit down to breakfast. Among the better class of patients I am in the habit of advising meat for breakfast; in addition, the patient can take milk, milk-cocoa, or milk with tea, but no coffee; he can have toast, Zwieback, and eggs.

If the patient's appetite is good and if he can eat a certain amount of meat for breakfast, he can get along without any further food until noon. In general it may be said that it is more rational in cases of this kind not to allow the patient to eat very frequently, for if food is ingested at frequent intervals, the gastric mucosa is irritated and the tone of the stomach consequently reduced; in many cases, however, it is necessary to feed the patient frequently, in part because these patients feel satiated even after a very small meal, in part because they have a desire for food at frequent intervals. Patients of this kind should eat a second breakfast at 10 o'clock in the morning, consisting of meat (ham, tongue) and a little toast.

Some patients complain of pain before dinner. This pain may be of two kinds : if the ectasy is severe and if the stomach has not succeeded in getting rid of the ingesta by this time, the pain may be caused by hyperchlorhydria ; in other cases again the secretion that is present in the stomach when it is empty irritates the mucous lining and causes the attack of pain. As a rule, the distress can be relieved by the administration of alkalis or by lavage. The latter procedure should be employed only in case of very violent pain or in those cases where remnants of food remain in the stomach.

At dinner the patient should, if possible, eat two kinds of meat. If there is no ectasy, soup may be given in addition to some meat dish, but only in moderate quantity ; if ectasy and atony are marked, as little fluid as possible should be introduced into the stomach. In emaciated individuals the necessary amount of fluid may be introduced by rectum.

Amylaceous food and dishes made of flour should be given in small quantities only.

If the patient has a frequent craving for food, a little milk-cocoa or tea with milk may be given in the afternoon. Before supper the stomach should be thoroughly washed out and the meal should consist of a small amount of meat and eggs.

This example may show how the diet should be regulated in moderate cases of continuous secretion of gastric juice. Numerous modifications are, of course, permissible to suit the peculiarities of each individual case.

The diet, at all events, should be as nourishing as possible and should not be bulky, in order not to put a severe strain on a stomach that is already atonic or at least has a tendency to atony. The amount of fluid and of amylaceous material should be limited ; gelatinous articles of food and fats may replace the latter. The diet should consist chiefly of proteid material ; milk is to be recommended in all cases.

If amylacea are administered at all, they should be given when there is no hydrochloric acid in the stomach ; they should further be administered in a finely distributed form, so that they cannot mechanically irritate the stomach. If possible, starches should be predigested before they are eaten. Soups and mushes made of oatmeal or aleuronat flour, aleuronat Zwieback, Maggi's flour, maltoleguminose, and similar articles of food are all to be recommended. Sugar solutions, particularly solutions of dextrose, are very good, for, in contradistinction to starchy preparations, they do not stimulate the hydrochloric acid secretion to any great degree ; they are also valuable in these cases because we know that in hypersecretion the conversion of starch into sugar is impeded and sometimes altogether arrested. In cases of motor insufficiency the administration of sugar solutions is, of course, contraindicated, because under these circumstances fermentation may occur. Honey may sometimes be tried.

All varieties of cabbage and the different varieties of turnips are to be excluded from the diet of these patients. Potatoes should be given in small quantities, and only mashed. Butter and cream are good, and are usually, though not always, well borne. It is best to avoid alcohol altogether, particularly strong alcoholic drinks. Spirituous liquors should be given only when their administration is especially indicated; wine may be given in the form of rectal enemata.

The articles of food enumerated include all those that are really indicated in these cases. The dietary will vary according to the presence or absence of dilatation; it is impossible to formulate any fixed dietary rules for this reason; any physician can readily construct a diet-list for himself. The caloric value of the different articles should, of course, be considered when this is done. I subjoin a diet-list that may serve as an example, and can refer those physicians who do not care to undertake the task of arranging diet-lists themselves to the work of Biedert and Langermann, where they will find a number of diet-lists.

DIET-LIST.

		Caloric value.
In the morning:	250 gm. of milk-cocoa, 3 Zwieback	385
At 10 o'clock:	100 gm. of raw ham, 20 gm. of toast	208
At noon:	Rice soup with the yolk of 1 egg	167
	200 gm. of beefsteak	272
	100 gm. of mashed potatoes	105
	70 gm. of poultry, 20 gm. of toast	208
In the afternoon:	250 gm. of milk-cocoa, 2 Zwieback	345
In the evening:	100 gm. of meat, 20 gm. of toast, 2 soft-boiled eggs	350
In addition, 60	gm. of butter in the course of the day	480
	Total	2520

This diet-list is indicated only in those cases in which there is not a high degree of ectasy; some authors object to it on the ground that proteid material is given in too abundant quantities; and many clinicians are at present advocating a vegetable diet; they claim that meat causes a greater secretion of hydrochloric acid. I should like to state, however, that, in the first place, it is not demonstrated that those articles of food that require much hydrochloric acid for digestion necessarily cause a greater secretion of gastric juice than those that do not require so much hydrochloric acid; in the second place, we know from experience that meat is well borne in cases of hypersecretion, whereas starchy food is not so well borne. This can readily be demonstrated by aspirating the stomach-contents after a meal consisting either of meat or of starchy food.

There are many other methods of treating cases of continuous secretion of gastric juice; above all, lavage of the stomach. All the measures advocated are intended to relieve the pain, to counteract the effects of ectasy, and to promote digestion.

No remedy is so efficient in relieving the attacks of pain in hypersecretion as a thorough lavage of the stomach. In some cases alkalis answer the purpose, or again a little milk or some hard-boiled egg or finely distributed egg-albumen will do the same. If the stomach contains remnants of food at supper-time, owing to the existence of motor insufficiency or ectasy, it is best to wash the stomach out before eating. The same will obtain in the treatment of these cases as in the treatment of motor insufficiency and ectasy; in order to avoid repetition, therefore, I refer to the paragraphs in which these conditions are discussed. If the stomach is thoroughly washed out before a new meal is introduced, an abnormal distention of the stomach is prevented and the ectasy in this way improved; on the other hand, lavage of the stomach performed at this time directly aids digestion. As soon as the acid residue is removed from the stomach, the organ can functionate like a normal stomach, with the exception, of course, that the hydrochloric acid will be increased at the height of digestion; if the stomach, therefore, does not contain free hydrochloric acid when the first food is introduced, it can digest a certain amount of starchy food for a time. The same conditions exist here as in hyperchlorhydria. In continuous secretion of gastric juice there is really no amylolytic stage of digestion; if lavage is performed, there is an amylolytic stage, although, of course, an abbreviated one. If the stomach is washed out as described, a certain amount of starchy food may unreservedly be given immediately after lavage, and this constitutes the chief advantage of this procedure. The physician will have to be governed by the intensity of the secretory function and the degree and the kind of ectasy in regard to the frequency with which he performs lavage; in many cases a single washing will suffice. The best time to administer this treatment is in the evening before supper; if ectasy is very severe, it is best to wash the stomach both morning and evening. I do not think that morning lavage alone is practical, because the stomach is only slightly relieved by this procedure, and favorable conditions for amylolysis are created only for breakfast. I do not think it is advisable to exclude amylaceous food altogether. I have repeatedly tried, in very obstinate cases, to nourish the patient for a time with a pure albuminous diet without any carbohydrates whatever; in every instance I was forced to modify these regulations because the patients soon lost all appetite, and because, in addition, certain peculiar symptoms appeared that resembled an intoxication. In the majority of cases lavage of the stomach before supper is all that is required. In the morning Carlsbad salts may be administered for the purpose of neutralizing any acid that may be present, and of causing its expulsion from the stomach. Only in those cases in which an abundant quantity of half-digested food is present in the morning, even though the stomach was carefully washed out in the evening, morning lavage may be instituted. The indication for this treatment is given more by the existence of ectasy of severe degree than by continuous secretion of gastric juice. It is well, in all cases of continuous secretion of gastric juice, to follow lavage by a thorough douching of the stomach with alkaline waters or with waters that contain some alkaline remedy. Some authors recommend douching the stomach with silver nitrate solutions, 1 : 1000, followed by a third irrigation of the stomach with lukewarm water. Penzoldt praises douching the stomach with boric acid (500 c.c. of boric acid solution of the strength of 10 : 1000).

He allows this wash to remain in the stomach for five minutes, and then thoroughly douches the organ. Other authors have recommended irrigation with bismuth, and I think this procedure is worthy of trial.

We can hardly get along without alkaline remedies; they do good at the height of digestion when hyperchlorhydria exists, and fulfil a useful purpose at this stage, namely, that of neutralizing the acid. They are further valuable if there is pure gastric juice in an empty stomach. The presence of an acid secretion under these circumstances usually causes pain and also prevents the solution and disassimilation of any starchy food that may be introduced. In these cases, alkaline remedies act only, however, on the acid that is present, and are capable of neutralizing only this. Alkalis should, therefore, on the one hand, be given at the height of digestion, or, on the other, before, or possibly together with, the meal. They should be given at the height of digestion if free hydrochloric acid is present at this time and causes pain; if, on the other hand, it is desired to improve amylolysis, they should be given before or together with the meal, not after it. If the introduction of large quantities of fluid is not contraindicated, alkalis may be given in the form of alkaline mineral waters. If there is severe ectasy, magnesia usta with bicarbonate of soda or ammoniomagnesium phosphate and similar remedies may be administered.

Many authors warn against the abuse of alkalis. I consider the administration of alkalis wrong if there is nothing in the stomach that is to be neutralized. According to the principles that we have enunciated, they should be administered only when this perversion must be corrected. A course of waters at Carlsbad has frequently been recommended. I never advise my patients to undergo such a course if ectasy exists, for I consider that alkaline remedies that are not administered in the form of waters are better for these cases. If the patient is suffering from simple hypersecretion without ectasy, such a course of waters may be permitted.

I can usually get along without narcotics. If the diet is adapted to each individual case; if the stomach is methodically washed out; if alkaline remedies are administered at the right time—we can usually dispense with narcotics. I hardly ever have to employ these drugs in my clinic where the patients can be carefully supervised and the diet exactly regulated. I administer morphin only in cases of extreme urgency, and even here I usually prefer the preparations of belladonna. I am not prepared to say whether the latter drug is capable of inhibiting gastric secretion; some authors, it appears, have advanced this view. [Since this was written Prof. Riegel has himself provided experimental evidence that atropin does reduce gastric secretion.—Ed.]

Massage of the stomach is never employed in the treatment of continuous secretion of gastric juice, but only in the treatment of ectasy that may exist at the same time. This procedure, as well as electric treatment of the stomach, is indicated only in the purely atonic forms of ectasy. Massage of the stomach may be combined with massage of the intestine in cases where constipation supervenes. If symptoms of ulcer are present, massage is, of course, contraindicated.

Strychnin given internally or administered subcutaneously has also been recommended as a remedy that is said to improve the tone of the stomach.

Laxatives proper are rarely necessary. It is usually possible to regulate the stools by the above-mentioned methods of treatment.

Other therapeutic measures are, of course, necessary under certain conditions. If, for instance, there is ectasy of high degree on the basis of a stenosis of the pylorus, the amount of fluid should be limited and the deficiency compensated by the rectal administration of fluid. The same applies to the treatment of ectasy in these cases as in cases in which ectasy is present without hypersecretion. Under certain extreme conditions rectal alimentation may even be indicated. All these methods are, however, directed more toward the treatment of severe degrees of ectasy than of hypersecretion.

If the thirst is very severe, small doses of Dover's powder or opium may be administered. If there is hemorrhage from the stomach, the same rules apply as in ulcer and hemorrhages from ulcer.

We know of no reliable remedy for the treatment of tetany. According to Kussmaul's theory, this complication is caused by the withdrawal of water from the tissues. For this reason Kussmaul recommends the administration of abundant quantities of fluid either by the rectum or in the form of subcutaneous infusions of salt water. From the point of view of the intoxication theory treatment of this kind seems justified; in practice, however, these methods have been found just as inefficient as the administration of narcotics and all the other methods that have been recommended.

In conclusion, a few words on the operative treatment of these cases. Continuous secretion of gastric juice of moderate degree, particularly if it is not complicated with ectasy or cicatricial stenosis, does not call for operative interference; if there is stenosis of the pylorus or if there is ectasy of high degree, an operative inroad may be advisable. Surgical measures are, of course, directed more toward the cure of the ectasy than of the hypersecretion. If a number of surgeons report cases in which hypersecretion disappeared immediately after gastro-enterostomy, we are not justified in concluding that this operation is to be recommended in the treatment of hypersecretion. I am of the opinion that such extreme measures should be employed only in those cases which exhibit ectasy and motor insufficiency of high degree. It is to be expected *a priori* that gastro-enterostomy will relieve not only the stagnation of ingesta, but also the stagnation of gastric secretion. The removal of stagnating stomach-contents is, however, not necessarily equivalent to the reduction of excessive secretion.

GASTRIC HEMORRHAGE.

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Etiology.—We can, of course, speak of gastric hemorrhage only when the blood ejected is derived from the stomach; blood from other organs, as the esophagus, the mouth, and the air-passages, may enter the stomach and be vomited, and this may simulate gastric hemorrhage. Vomiting of blood, therefore, cannot be considered a proof of gastric hemorrhage. On the other hand, vomiting of blood may be absent in a large proportion of cases of gastric hemorrhage.

If hemorrhage occurs in the stomach, we must conclude that the normal relations between the pressure exercised on the vessel-wall and their natural powers of resistance is disturbed; this applies to all hemorrhages.

The resisting powers of the blood-vessel walls may be reduced or lost in some one place, or the pressure exercised from within may be increased. In the majority of cases hemorrhage is due to the former cause, so that diapedesis of blood, in other words, hemorrhage, rarely occurs unless some coarse lesion of the vessel-wall exists. Even a considerable increase in the blood-pressure can rarely produce oozing of blood from normal arteries. In the veins and capillaries increased blood-pressure may, however, force blood through the vessel-wall or cause rupture.

Gastric hemorrhages may be due either to some injury sustained from without or some process that develops within the stomach. The former group includes direct injuries to the mucous lining of the stomach that may be either mechanical or chemical. Foreign bodies that are swallowed, pieces of bone, needles, etc., injure the stomach mechanically. The hemorrhages that occasionally follow the introduction of the stomach-tube are also to be attributed to mechanical injury of the gastric mucosa.

Corroding substances—mineral acids, caustic alkalis, and similar bodies—may cause hemorrhage by chemical injury to the stomach; these chemicals are swallowed either for suicidal purposes or by mistake in place of drugs.

The hydrochloric acid of the gastric secretion may, under certain circumstances, erode blood-vessels; this cannot occur, however, as long as the mucous lining of the stomach is perfectly normal, but it can occur whenever the circulation is interfered with or arrested, when there is extravasation of blood, thrombosis, or embolism in one or the other of the larger divisions of the gastric circulation.

The last-named class of cases really belongs to the second group, which includes all the processes that can lead to hemorrhage and that occur within the stomach itself. Here the injury is not sustained from without, but the mucous membrane itself is changed.

In addition to the thrombotic and embolic processes that we have mentioned, varicose changes in the veins, aneurysms, atheroma of the arteries, fatty degeneration of the vessel-wall, etc., may lead to hemorrhage.

Gastric hemorrhage has also been seen to follow a blow in the stomach region or a fall, causing concussion of the gastric region. In some of these cases rupture of a vessel was caused directly by such an accident; in others there was a more or less severe degree of blood extravasation, followed secondarily by the erosion of the blood-vessel.

Another prolific source of gastric hemorrhage is venous stasis, particularly in the portal area. In pylephlebitis, cirrhosis of the liver, and tumors of the liver, gastric hemorrhage may occur. Compression of the lower vena cava by tumors above the entrance of the hepatic veins may cause hemorrhage into the stomach. Stasis in the vena cava following lesions of the heart or the lungs less frequently leads to gastric hemorrhage.

[The appearance of hematemesis following hepatic cirrhosis is often unexpected, as such cases are unusually free from the commonly recognized symptoms of portal obstruction.

Preble[1] has collected 60 cases of fatal hemorrhage due to cirrhosis of the liver in which he found esophageal varices present in 80 per cent., and in more than one-half of these microscopic ruptures could be detected. Fatal hemorrhages occurred where no esophageal varices were discovered, the blood in this class of cases escaping from the gastro-intestinal mucous membrane. Only 6 per cent. of the 60 cases showed the ordinary clinical evidences of cirrhosis—that is to say, in 92 per cent. ascites, enlarged spleen, and distended superficial abdominal veins were absent. It is interesting to note that there is frequently absence of ascites, although the cirrhosis may be far advanced. This apparently results from the free anastomosis of the portal with the systemic veins, and is well illustrated in the cases reported by Stockton.[2] Curschmann recounts a somewhat similar experience.[3] Of 13 patients who died from profuse hemorrhage with cirrhosis of the liver, 12 exhibited esophageal varices, and in but one fatal case was the hemorrhage found to depend upon a varix in the walls of the stomach. I agree with Curschmann that these hemorrhages are apt to be very severe, and that it is rare to find what some authors mention as commonly happening, namely, small repeated hemorrhages. Curschmann says that the simple regurgitation of blood without effort to vomit points strongly to hemorrhage from the esophagus. This rule, however, is not of much value, for the reason that the regurgitation rarely occurs, the vomiting exhibiting the same character as when the hemorrhage occurs inside the stomach itself.—Ed.]

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[1] *Amer. Jour. Med. Sciences*, March, 1900.

[2] *Jour. Amer. Med. Assoc.*, Sept. 28, 1891.

[3] *Deutsch. med. Wochenschr.*, April 17, 1902.

In certain constitutional diseases, in severe forms of anemia, in leukemia, in Hodgkin's disease, in hemophilia, in scurvy, in morbus maculosus, hemorrhage occurs into the stomach as into other organs. That form of gastric and intestinal hemorrhage called melena, occasionally seen in the new-born, belongs to this group.

It would lead us too far were we to discuss the causes of gastric hemorrhage in the first-named group of constitutional disturbances. I will limit myself to a few remarks in regard to melena. The term melena is employed not so much to designate a definite anatomic disease, but merely a group of symptoms. We speak of melena if a child loses considerable quantities of blood either by vomiting or in the stools, or in both ways, in the first few days after birth. The cause of this hemorrhage may vary greatly: in some cases we are dealing with an infection; in others, with hemophilia; in still others, with scurvy. Ulcers of the stomach or of the intestine have also been found in a very few cases.

Landau[1] assumes that these gastro-intestinal ulcers are not of intra-uterine origin, but are caused by the entrance of thrombi from the umbilical vein and the ductus Botalli into the arteries of the stomach or intestine. Other authors again assume that this condition is caused by bacterial influences.

The disease-picture is as follows: Bloody stools, or, less frequently, bloody vomiting, are seen usually on the second day or a few days later. These hemorrhages recur at frequent intervals, and in severe cases lead to the death of the infant in a short time; in milder cases they gradually stop and recovery finally occurs.

The menstrual type has also occasionally been seen in gastric hemorrhages. In some forms of hemorrhage from the stomach that originate from an ulcer the bleeding has been seen to coincide with the menstrual period. In cases of amenorrhea periodic gastric hemorrhage is occasionally observed. I do not think that we are justified in calling this form of hemorrhage vicarious, although many authors elect to do so. A gastric hemorrhage can, in my opinion, hardly be considered a substitute for the physiologic process of menstruation.

Schiff, Brown-Séquard, Ebstein, and Ewald have performed a number of experiments that have become classic; they show that injury to certain parts of the brain, separation of the peduncles of the brain, of the optic thalami, of the corpus striatum, of the crura cerebri, of the anterior corpora quadrigemina, and also of certain portions of the spinal cord, can lead to hemorrhage, bloody infiltration, and ulceration of the stomach-walls. All this makes it probable that under certain conditions gastric hemorrhage may originate from lesions of the central nervous system. So far no clinical observations have been made that demonstrate that these results that follow artificially produced nervous lesions in animals have their counterpart in man.

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[1] Landau, *Ueber Melæna der Neugeborenen*, Breslau, 1874.

A number of cases of hysteria have been reported in which hematemesis occurred either during a hysteric attack or between the attacks; it is claimed that hysteric hemorrhages of this kind are characterized by the frequency with which they occur. As the amount of blood lost is very small, the general health of the patient is not impaired.

The most frequent causes of gastric hemorrhage are round ulcer of the stomach and carcinoma; then, in their order of frequency, mechanical disturbances of the circulation, intoxications, and direct injuries by foreign bodies.

Gastric hemorrhages, for reasons that we can well understand, occur chiefly in youth and in the middle years of life; more frequently in women than in men; in old age they are frequently seen in sufferers from carcinoma.

Pathologic Anatomy.—Hemorrhage from the stomach, it may be stated at once, is rarely fatal. Wherever death ensued, the autopsy revealed the picture of extreme grades of anemia: all the organs were very pale and contained a minimal quantity of blood.

In the stomach considerable quantities of fluid or coagulated blood are usually found. In some instances the stomach was even found dilated and distended by blood.

The appearance of the blood varies: it may be either light-red or blackish. The changes that occur in the blood after gastric hemorrhage will depend on the acidity of the stomach and the length of time that the blood remains in the stomach.

The source of the hemorrhage may be found to be a small hemorrhagic ulceration or an ulcerated carcinoma; in other cases a round ulcer or a ruptured aneurysm of one of the vessels of the gastric mucosa. In a few rare cases rupture of a varicose vein caused hemorrhage. Lancaster has recently reported a case in which a woman of thirty-three years who had been perfectly healthy all her life suddenly had a violent gastric hemorrhage. Hematemesis recurred several times, and led to the death of the patient in a short time. The cause of the hemorrhage in this case was found to be a varicose degeneration of several branches of the gastro-epiploic vein in the mesentery and in the submucosa of the stomach. The largest of these varices in the gastric mucosa was ruptured, the opening being about as large as a pin-head.

In many cases it is possible to discover a coagulate in the wall of some eroded blood-vessel; in this case, of course, the source of the hemorrhage is immediately revealed. In other cases, particularly in capillary hemorrhages, the primary cause of the hemorrhage frequently avoids detection. In many cases, moreover, it must not be forgotten that hemorrhage does not originate in the stomach, but blood from the nasal cavities, the mouth, or the esophagus secondarily enters the stomach.

Symptoms.—The most important and typical symptoms of gastric hemorrhage are bloody stools and bloody vomiting. Many cases of gastric hemorrhage are never recognized; this applies particularly to those slight degrees of hemorrhage that do not cause vomiting and where so little blood is shed that the stools are not visibly changed. In addition, in these milder cases the general health of the patient is not visibly affected.

But even large hemorrhages may be overlooked in cases where the blood is voided by the rectum. As a rule, the patients do not inspect the stool, and even physicians frequently do not attach enough importance to the inspection of the dejecta. The physician should not omit an examination of the stools in all cases where great pallor and weakness appear suddenly; he should not only do this in cases that show symptoms of disease of the stomach, but also in cases that never develop any such symptoms; provided, of course, that the sudden weakness cannot be explained by other conditions. Every physician will probably remember cases of some mild and insignificant gastric trouble in which the patient suddenly turned very pale and became very weak, and in which the explanation of these symptoms was not furnished until the next rectal passage was examined.

The first task must be to determine whether a hemorrhage has occurred; in the second place we must determine whether the blood comes from the stomach. If these two problems are solved, the special form of stomach-disease can also, as a rule, be diagnosed.

I cannot, of course, in this place discuss all the possible symptoms that might precede an attack of hematemesis: to do this I would have to repeat the whole symptomatology of ulcer, carcinoma, and other diseases of the stomach. I will limit myself, therefore, to explaining those symptoms that are caused directly by hematemesis.

The first symptoms are the well-known signs of internal hemorrhage; the greater the loss of blood, the more pronounced these symptoms. They may vary according to the rapidity with which the blood is poured into the stomach; if it is shed rapidly, the patient suddenly faints and falls unconscious, and may even develop convulsions; if the hemorrhage is slight, these symptoms are absent, but the patients complain of increasing weakness. Many patients state that they have a feeling of filling up and of heat in the stomach during the hemorrhage. Hematemesis is rarely ushered in by a sudden attack of pain.

Whether a hemorrhage of this kind occurs rapidly or slowly, the picture of a more or less pronounced acute anemia will always appear, provided the hemorrhage is sufficiently severe. The patients look very pale, feel extremely weak, can hardly sit up in bed, faint easily, complain of vertigo, buzzing in the ears, blurring of sight. If the hemorrhage is very severe, nausea and vomiting usually occur.

The quantity of blood that is vomited varies greatly: sometimes there are only one or a few tablespoonfuls; in other cases from one-half to one liter and more; abundant quantities may be vomited repeatedly at short intervals. When vomiting of blood occurs, the bowel-passage should be inspected, for a portion of the blood usually leaves the body in this way.

The appearance of the vomited blood also varies: it may be arterial or venous; much will depend on the quantity of blood and the time it remains in the stomach, the constitution of the gastric juice, and the presence or absence of food-particles in the stomach. If vomiting occurs soon after the hemorrhage, and if no particles of food are contained in the vomit, it can readily be seen that the material raised is really blood; but if the blood remains in the stomach for a long time, the action of the gastric juice soon converts it into a dark chocolate or coffee-ground color.

It frequently happens that coffee-ground-colored masses are aspirated from the stomach together with food-particles, particularly in cases of ectasy in which no symptom even remotely suggested gastric hemorrhage. This is seen particularly in carcinoma.

In a number of cases, as we have already said, gastric hemorrhage does not lead to the vomiting of blood, but the whole quantity of blood passes into the intestine and is deposited with the stools; large quantities of blood in the stools are readily recognized by the dark, almost black, color that they impart to the dejecta. If the quantity is small, simple inspection of the stools may fail to reveal the presence of blood, and other methods must be employed to determine its presence.

Quite frequently a rise of temperature will be seen after a severe hemorrhage. Some authors even speak of an anemic fever caused by loss of blood. It is known that a rise of temperature of this kind can be experimentally produced in animals by withdrawing large quantities of blood; whether or not to call this pyrexia fever is a question that cannot be decided in the light of our present knowledge on the nature of the fever. This rise of temperature, at all events, is no peculiarity of hemorrhage of the stomach, as it is seen in hemorrhages into all other organs. I need only mention the well-known fact that fever occurs after blood-letting. Leichtenstern has made the statement that gastric hemorrhage caused by simple ulcer is usually accompanied by a slight or by a fairly high degree of fever, and that this is a frequent, in fact, a regular, symptom of this condition. I cannot agree with this statement, and I do not think that this fever is at all peculiar to gastric hemorrhage occurring in ulcer. If we see this rise of temperature more frequently in ulcer than in any other lesion of the stomach, this is undoubtedly due to the fact that ulcer leads to hemorrhage more easily than any other affection of the stomach. To judge from my personal experience, the rise of temperature—that is, the fever,—depends on two factors: on the one hand, on the rapidity with which the hemorrhage occurs; and, on the other hand, on the amount of blood that is shed. In small hemorrhages I have never seen a rise of temperature.

It is not established how this rise of temperature originates. The term anemic fever designates nothing more than that fever may occur after hemorrhages that lead to anemia. Some authors claim that the fever is caused by the resorption of toxic products from the intestine, and that these poisons are generated by the putrefaction of the blood; others assume that bacteria enter the circulation through the eroded blood-vessels; still others claim that the fever is due to irritation of the heat-centers by the impoverished blood.

The most satisfactory explanation is a purely mechanical one. If the anemia is severe, the internal organs become congested and the peripheral parts of the body grow anemic and cooler, and the blood circulates less rapidly through these parts; in this way the radiation of heat is diminished and the internal heat rises. The pyrexia observed under these conditions greatly resembles the postmortem rise of temperature. Whatever explanation is accepted for the phenomenon, we must remember the fact that in severe gastric hemorrhages a rise of temperature occurs that may last for several days.

The pulse, as a rule, is greatly accelerated; occasionally it cannot be felt at all, so that the beats must be controlled by palpation of the heart. In contradistinction to the small and hardly perceptible pulsations of the radial artery, the apex-beat may be very much stronger than normal.

Rare sequelæ of violent hemorrhages are delirium and visual disturbances—amblyopia and amaurosis. As a rule, these visual disturbances, which are frequently accompanied by fainting spells in cases of severe hemorrhage, soon disappear; in some instances, however, permanent blindness in one or both eyes may result; the cause of this is an atrophy of the optic nerve. Ziegler, in cases of this kind, has discovered foci of fatty degeneration in the optic nerve and in the retina. It has not been determined why this occurs; Ziegler assumes that the primary cause is local vessel-contraction; others assume that there is hemorrhage into the optic nerve, and still others (Förster) claim that it originates from a serous imbibition of the retina with extravasation.

In the majority of cases the patients recover quickly, provided the loss of blood is not too great. In more severe cases anemia may persist for a long time. The subsequent course of the disease will naturally depend on the primary trouble that caused the hemorrhage. Cases in which gastric hemorrhage is so severe as to cause death are rare.

Diagnosis.—The diagnosis of gastric hemorrhage is, in the majority of cases, easy. If hematemesis and bloody stools are seen in a subject that is suffering from ulcer and carcinoma, the diagnosis of gastric hemorrhage can usually be made. In other cases again, particularly where the hemorrhage is slight or where the blood remains in the stomach and intestine for a long time, it is often difficult to decide whether the masses that are found in the vomit and the stool really consist of blood. We have a number of methods to determine this point: if the presence of red blood-corpuscles can be revealed by microscopic examination, the diagnosis is, of course, easy; if the blood, however, remains in the stomach for a considerable length of time, the red blood-corpuscles are destroyed and cannot be discovered in the vomit nor in the feces. Only a positive finding is of value in rendering a decision; a negative finding demonstrates nothing.

Teichmann's test, if positive, is conclusive. It consists in the microchemical formation of crystals of hemin. As almost minimal quantities of material are used for making this test, the method is not altogether satisfactory; blood may be present in the stools and still avoid detection by this method.

Heller's test—addition of potassium hydrate and boiling—is very simple, but the results are not absolutely conclusive because other pigments occur in the stomach-contents and the feces that give the same reaction.

The spectroscopic method is altogether reliable, but, on the one hand, it is too complicated, and, on the other, it requires an instrument that every physician does not possess.

A reliable method and one that can easily be executed is van Deen's test as modified by Weber. The stomach-contents or a watery suspension of fecal matter is mixed with one-third volume of glacial acetic acid and shaken with ether. As soon as the two fluids separate and the upper ethereal layer has become clear, a few cubic centimeters of this acid ethereal extract are poured off and mixed with about 10 drops of tincture of guaiac and 20 to 30 drops of turpentine. If blood is present, the mixture turns bluish-violet; if it is not present, it turns reddish-brown, frequently with a slight greenish tinge. A still better procedure is to remove the blue coloring-matter from the mixture by shaking with chloroform. In interpreting the findings in this test it must, of course, be determined whether or not the patient ate raw or half-raw meat a short time before, as blood may be present in the stools from this source.

If the presence of blood in the stools or the vomit has been demonstrated by one of these methods, the second point to be decided is whether the blood really comes from the stomach or whether it comes from some other organ.

Blood from the nose, the air-passages, or the mouth may be swallowed and later vomited. In general, however, we may succeed in discovering the source of these hemorrhages from the history of the case, the course of the disease, and a careful objective examination of the patient.

Many clinicians claim that there is great difficulty in making a differential diagnosis between hematemesis and hemoptysis. It is true that when blood is vomited some of it may be aspirated into the air-passages and later be expectorated during coughing efforts, and inversely that a part of the blood that comes from the air-passages or the mouth may be swallowed and later vomited. A careful anamnesis will usually reveal the true condition of affairs. Frequently the statements of the patient are very unreliable, as they do not know themselves whether they vomited the blood or coughed it up.

Nevertheless, we may usually succeed in making a differential diagnosis between hemoptysis and hematemesis. In the former instance the history will, as a rule, show that catarrh or other disturbances of the respiratory passages existed for some time previous to the accident; it will also show that at the beginning of the attack there were coughing and tickling in the throat and that gagging and vomiting came later. In the former case, moreover, the blood is light red, foamy, is mixed with mucus, and has an alkaline reaction, and bloody sputum is expectorated for several days. A careful examination of the apices of the lung will also usually reveal other catarrhal phenomena or deep-seated lesions, whereas an examination of the stomach will fail to reveal any abnormal conditions.

The fever that usually occurs is less important, for this may be caused by the hemorrhage alone; if fever was present before the attack, it may aid in the diagnosis.

If the blood, on the other hand, comes from the stomach,—that is, if there is hematemesis,—the anamnesis will usually show that dyspeptic symptoms were complained of for a long time before the attack of hemorrhage occurred. The attack itself generally begins with a feeling of oppression and fulness in the region of the stomach, followed, as a rule, by nausea and vomiting. Coughing commonly occurs later. In contradistinction to the blood raised in hemoptysis, the blood in hematemesis is, as a rule, very likely to be dark, only rarely light red; it is usually mixed with particles of food. Bloody vomit is acid. Examination of the lungs reveals nothing abnormal, whereas examination of the stomach region frequently indicates disturbed gastric function. In other cases certain indications will point to some disease of the liver and disturbances in the portal circulation or other factors. There is no expectoration, and even though a small quantity of bloody expectoration is raised in the beginning, it is not seen later on. The stools usually appear black for some time after the hemorrhage.

With the aid of all these points the decision whether we are dealing with a case of hematemesis or hemoptysis can usually be rendered.

In doubtful cases the sputum may be examined for tubercle bacilli. The differential diagnosis will be difficult only in those rare cases in which there are conditions present both in the lungs and the stomach that might favor hemorrhage.

A careful inspection of the mouth and the gums will readily show whether or not the blood could have come from these parts; on the other hand, it is frequently almost impossible to differentiate esophageal hemorrhages from gastric hemorrhages. I do not refer, of course, to those cases in which there is pronounced evidence of some esophageal lesion, but to those cases in which there is, for instance, a varix of the esophagus that produces no symptoms until a violent hemorrhage occurs.

It is impossible without the most careful examination to determine whether the blood comes from an ulcer, a hemorrhagic erosion, a carcinoma of the stomach, or some other lesion of the organ.

Prognosis.—The prognosis of gastric hemorrhage is dependent on the amount of blood lost. In this place, of course, we cannot speak of the prognosis of the primary disease leading to the hemorrhage. In regard to this we must refer to our discussion on the different diseases of the stomach that can lead to hematemesis.

Small hemorrhages *per se* are without danger. They are important because there is always danger of a recurrence; even large hemorrhages, as a rule, do not directly endanger the life of the patient. The most violent hemorrhages are seen in ulcer, but even these usually terminate favorably even though there be a colossal loss of blood leading to fainting spells, collapse, apparent loss of pulsation, etc.; as a rule, the patients recover. I have, however, seen cases of profuse hemorrhage that terminated in death so rapidly that it was impossible to perform transfusion or infusion. In other cases, again, although the patients were in the agonal stage, energetic procedures warded off the immediate danger.

Treatment.—We cannot enter into a discussion of the prophylaxis of hemorrhages of the stomach in this place, as the measures employed for this purpose coincide with the treatment of the primary disease. Whenever it is possible to cure or to relieve the primary disease, the danger of hemorrhage will be correspondingly decreased or will be altogether avoided. In this sense the rational treatment of an ulcer of the stomach is to a certain degree the best prophylactic against hemorrhages. In the case of carcinoma the methods that we can employ are not so satisfactory, for we know of no method that will stop the progress of the disease and the disintegration of the neoplasm.

As soon as hemorrhage occurs, the first task of the physician is to place the diseased organ completely at rest. The patient should be ordered to lie perfectly still in a horizontal position, and should not be allowed to change his position even for the purpose of defecating or for passing urine.

An ice-bag should be applied to the region of the stomach. In order to avoid too much pressure by the ice-bag, a hoop should be placed over the epigastric region and the ice-bag suspended from it. All excitement should be avoided. The physician should attempt to calm the patients, as they are usually very much frightened and excited, and should assure them that the hemorrhage is completely without danger. In order to give the diseased stomach a complete rest, all irritation of the organ should be avoided; the patient should take no food or drink whatever by mouth; all that he may be permitted to swallow is cracked ice; this quenches the thirst very satisfactorily; it should not, however, be swallowed until it has melted in the mouth. If it is necessary to administer nourishment, it should be given by the rectum; as a rule, however, it is altogether unnecessary to feed the patient, at least during the first few days. If the loss of blood is very severe, fluid may be introduced by the rectum. In these cases enemata of salt should be given, or, if it is desired to stimulate the patient at the same time, enemata of wine and bouillon (one-third wine, two-thirds meat-broth).

It is not only unnecessary, but even harmful, to administer internal remedies in severe hemorrhage of the stomach, for, in the first place, they cannot possibly affect the bleeding spot itself, and, on the other hand, they stimulate gastric secretion and the motor functions of the stomach. It should be one of our chief purposes to avoid these very things. If it is desired to give some drug for the hemorrhage, a subcutaneous injection of ergotin may be administered. If there is much cardiac weakness or if it is desired to prevent fainting spells, camphor, ether, or camphor-ether may be injected subcutaneously.

If the life of the patient is directly endangered as the result of the great loss of blood, subcutaneous or intravenous injection of salt solutions should be practised. The general practitioner will probably prefer the former method, because it does not require an assistant and can be performed with a few simple appliances that can be procured anywhere; all that is needed is a hollow needle, a rubber tube, and a glass funnel or irrigator. A 0.6 to 0.75 per cent. salt solution should be employed.

According to my experience, the method of subcutaneous injection is preferable; it is simpler, more rapid, and less dangerous; the only objection to it is that the absorption of the water injected may be difficult in cases of great heart-weakness. This objection, it appears to me, is only theoretic, however; in those cases that I have observed the salt solution that I injected subcutaneously was rapidly absorbed, even though the heart action was very much reduced. It is well in these cases to massage the parts, as this aids the absorption of the injected fluid.

[In the treatment of hematemesis some benefit has been derived from the introduction of a solution of adrenalin chlorid, 20 to 30 drops of a 1 : 1000 solution given in one-half ounce of water every hour. The hypodermic injection of the same substance in a somewhat similar dose is theoretically of value, and has occasionally given good results. If the hemorrhage is due to erosion, the taking of about 200 c.c. of a 10 per cent. solution of gelatin, to be repeated two or three times a day, as recommended by Poliakow,[1] should be tried, and this especially applies to cases in which the hemorrhage is prolonged. In the acute cases it is best to allow nothing to be swallowed; even a small amount of water, or pellets of ice, should be avoided. The use of hypodermic injections of morphin to induce mental and physical quietude is good practice. Whether it is best to use iced water or hot water by the rectum has been considerably discussed, and the question is not yet settled. Enemata of normal salt solution are useful in restoring the strength of the patient and in relieving thirst, but it is unwise to resort to this too quickly unless the patient is *in extremis*, for the reason that the increased blood-pressure may reëstablish the hemorrhage that has ceased. It is advisable to give a small amount and to repeat this frequently rather than to use half a liter or a liter at one time. There is considerable evidence adduced to show that, contrary to the views once held by Winternitz, nutritive enemata rather lessen the acidity of gastric juice, and may, therefore, be safely recommended after hemorrhage from gastric ulcer.—Ed.]

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[1] *Lyon. Méd.*, 1898, No. 38.

MOTOR INSUFFICIENCY AND ECTASY OF THE STOMACH.

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See also the well-known works of Boas, Ewald, Rosenheim, Bouveret, Fleischer, Wegele, Fleiner, and others, on stomach-diseases.

Introductory Remarks.—Disturbance of the motor powers of the stomach in the sense of inefficient work of the stomach in this direction is frequently encountered. The condition may be seen either together with other diseases of the stomach or may appear as an independent disease. Even though it is no disease *sui generis*, it can produce a certain number of characteristic symptoms and may call for definite therapeutic measures; consequently it merits special discussion.

In no field of stomach pathology is there such a diversity of opinion as in the field of the perversions of motility. Some authors make a clear distinction between atony and motor insufficiency; others consider the two conditions identical; some distinguish motor insufficiency from ectasy, and still others wish to eliminate the old term ectasy altogether from the nomenclature of stomach-diseases. I do not think that this is justified. The condition that the older physicians called ectasy is encountered to-day and represents a distinctive disease-picture. Modern research and investigation have taught us to distinguish the milder degrees from the more advanced forms of motor insufficiency, whereas the older physicians, who did not possess our modern adjuvants, recognized only those more severe degrees of motor insufficiency that were combined with dilatation.

Physicians have always noticed cases in which the stomach was extremely dilated, so that it appeared like a flaccid sac lying in the abdominal cavity and was incapable of propelling its contents into the intestine. Patients afflicted in this way usually suffered from attacks of vomiting; the stomach, so to say, would run over and attempt to get rid of its contents by vomiting; as soon as this occurred it decreased in size. A number of cases of this kind are reported in the older literature, and every physician of experience has probably observed similar ones. Nowadays we rarely encounter severe cases of this character, certainly less frequently than we did twenty-five years ago. This is due, above all, to the general introduction of the stomach-pump into practice; Kussmaul[1] deserves credit for having advised the employment of the stomach-tube for therapeutic purposes in the treatment of dilatation of the stomach.

The older physicians used the term dilatation of the stomach not only to designate that the organ was abnormally distended, but also that the stomach was unable to get rid of its contents within the normal time. In some of the works of the older authors we find particular emphasis laid on the flaccidity of the organ. Bonet (1679), for instance, in describing a dilated stomach, says that "it is flaccid and white, like softened paper, has no folds, and reaches as far as the symphysis."

The physicians of former days were probably not familiar with the condition that we call "megalogastria" nowadays. With this term we designate an abnormally large stomach that, however, performs its functions in a normal manner. The sense of the terms "ectasy, dilatation of the stomach," is that the stomach is merely larger than normal; in practice, however, we are accustomed to call those cases ectatic in which the stomach is not only abnormally large, but in which there is, at the same time, motor insufficiency, so that the stomach is permanently overfilled and abnormally distended. In this latter sense, therefore, we can still employ the word "ectasy" to-day.

As I have repeatedly stated, our chief duty at the bedside is to determine the character of the functional perversions existing; it is not sufficient to discover the anatomic basis of the disease. This does not imply that we should neglect the study of the anatomic changes that may exist. For this reason it is important to find out whether the stomach is enlarged or not in cases in which the motor functions of the organs are perverted.

From a practical point of view, moreover, the degree of motor power of the stomach is very important; as soon as motor insufficiency obtains, the stomach may either become enlarged or it may not. The stomach, on the other hand, may be abnormally large or very much distended, and still possess normal motor powers; and, again, the stomach may be of normal size and still be insufficient in its motor powers; in fact, it may be reduced in size and still insufficient in the motor sphere, particularly in cases of diffuse cirrhosis and in cancerous lesions that infiltrate the stomach-walls over large areas.

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[1] *Deutsch. Arch. f. klin. Med.*, 1869, vol. vii.

Ectasy and motor insufficiency, are, therefore, two distinct conditions, and although severe degrees of motor insufficiency may be frequently associated with dilatation, we cannot, nevertheless, eliminate the term ectasy from our nomenclature. For these reasons I cannot agree with Boas, who has done particularly good work in the study of motor insufficiency, when he says that "the word ectasy, or dilatation of the stomach, should be dropped, because its employment is not justifiable in the light of our present knowledge."

Ectasy means dilatation, permanent enlargement of the stomach combined with motor insufficiency.

The term "motor insufficiency" alone merely signifies that a perversion of function exists in the sense that the motor powers are insufficient.

This condition may originate in different ways: it may be caused by mechanical obstructions; it may be due to a congenital or an acquired muscular weakness of the stomach-walls. In the latter case we speak of atony. Atony signifies relaxation; it is not correct, therefore, to consider atony and motor insufficiency as identical, although many authors do this. In stenosis of the pylorus the motor powers of the stomach are insufficient—that is, the motor power of the stomach is not sufficient to propel the ingesta from the organ within the normal time. This does not mean that atony is present; on the contrary, the musculature in many cases is hypertrophic, but, nevertheless, incapable of performing its normal tasks, and is consequently insufficient. Atony is used to determine a simple condition, whereas motor insufficiency includes much more; atony, in other words, is only a particular form of motor insufficiency produced by relaxation of the muscularis.

Motor insufficiency, whether it is due to atony or whether it is accompanied by hypertrophy of the muscularis, may lead to dilatation of the stomach, but does not necessarily do this. We cannot speak of ectasy until the motor insufficiency has progressed to such a degree that the organ has become enlarged. A simple enlargement of the organ does not necessarily constitute an ectasy.

Large stomach, so-called "megalogastria," is always found by chance, for it causes no symptoms. This condition does not possess the most characteristic feature of ectasy—namely, the constant overfilling of the stomach and the abnormal retention of ingesta. No physician probably has ever used the term gastrectasy to designate a condition in which the stomach could always get rid of its contents within the normal time. A stomach that is simply enlarged can usually be distended in an abnormal way when it is inflated with carbonic-acid gas or air; in other words, it is abnormally large, but nevertheless it is capable of performing its functions and of getting rid of the ingesta within the normal time.

Abnormal enlargement of the stomach may be either congenital or acquired. Many persons who eat very much, particularly those who live on a vegetarian diet, acquire an abnormally large and an abnormally distensible stomach. It is true that a subject with an acquired large stomach is more predisposed to atony than a subject in whom this condition does not exist. As long, however, as the stomach is capable of performing its functions within the normal time, we cannot speak of ectasy in the clinical sense.

If, therefore, we adhere to the term "ectasy" in the sense in which it has always heretofore been employed, I see no reason why we should omit this word from our nomenclature. In a clinical sense, as I have said, we speak of ectasy wherever there is permanent dilatation with motor insufficiency of the stomach.

This motor insufficiency, however, does not necessarily imply that there is atony in the proper sense of the word; ectasy may be accompanied by a true reduction in the muscular powers of the organ: then we speak of atonic ectasy; or, on the other hand, it may be accompanied by an increase in this muscular power: then we speak of hypertonicity. Cases of this kind are encountered particularly when some obstacle is presented to the exit of ingesta through the pylorus, as in stenoses of the pylorus. This condition may be differentiated from the first form by calling it hypertonic ectasy.

The term, "motor insufficiency," therefore, merely signifies that there is a perversion of the physiologic functions—a functional insufficiency of the stomach. When we use the term ectasy, we imply that there are, at the same time, an anatomic change and enlargement of the organ and that the motor powers may be absolutely or relatively insufficient. Megalogastria signifies an enlargement of the organ in which the motor powers are still normal.

In practice these conditions can readily be differentiated. It is natural, of course, that there should be different degrees of motor insufficiency—severe degrees and mild ones. The severe degrees, as we might expect, are frequently accompanied by dilatation. The fact that motor insufficiency of the second degree, as modern authors call it, may frequently be accompanied with dilatation, so that a clinical picture is presented that corresponds to the symptom-complex of gastrectasy of the older authors, does not justify us in dropping the word ectasy. This is apparent, for there are, in fact, cases on record in which there was motor insufficiency of high degree and in which the size of the organ was not increased.

I believe, therefore, that we should differentiate motor insufficiency from ectasy. It is true that a sharp distinction cannot be drawn, and it is hard to designate the point at which we shall begin to speak of an enlargement of the organ, for the boundaries of the normal organ fluctuate within wide limits; as a matter of fact, they vary almost every hour. But we are not attempting to determine the first stages of an enlargement of the stomach, but only the highly developed degrees of this condition. Nowadays this is not difficult.

In the following paragraphs I will discuss ectasy and motor insufficiency separately. By doing this I depart from the ordinary method of subdivision that is based on anatomic findings. I will not modify this new departure any further, but will refer to what I have said in the introduction. For the present we are unable to differentiate the different diseases of the stomach from an anatomic point of view alone. There are a number of functional diseases of the stomach that have no anatomic basis proper; nevertheless, they present a well-rounded clinical syndrome. Among these perversions we have motor insufficiency whether or not this condition is accompanied by dilatation of the stomach. Here we are always dealing with a perversion of function, due in its ultimate consequences to the fact that the normal relation between the propulsive power of the stomach and the amount of work to be done is disturbed. This perversion may be due to a variety of causes. The propulsive power may either be reduced or the amount of work to be done increased. If we call all these cases motor insufficiency, we do this because the ultimate result is the same—that is, the work is not done in the normal way and within the normal time. In one case we may be dealing with an absolute primary loss of muscle power: then we speak of atony; in another case the muscular power may be abnormally increased,—there may even be hypertonicity,—but the amount of work to be done be so large that even this increased muscle power becomes insufficient. The ultimate effect of either condition will be the same whether we are dealing with a primary atony or a direct loss of motor power.

Ectasy may, in the same sense, be two-fold in origin: it may be either atonic or hypertonic. To the latter class belong those cases in which there is an obstruction in the region of the pylorus, so that the ingesta cannot be propelled from the stomach within a normal time. Here we see hypertrophy and dilatation of those portions of the stomach that are situated behind the obstacle; there will be hypertrophy of the muscularis, and at the same time dilatation of the stomach. This, perhaps, is analogous to the conditions we see in the heart in stenosis of one of the valves. In these cases, then, we see ectasy combined with hypertrophy and increased muscular activity, in contradistinction to the other cases of atonic ectasy. This hypertrophy, however, is not sufficient to reëstablish normal conditions.

The main task in the diagnosis of these cases is not only to determine the existence of motor insufficiency and ectasy, but also to discover from what these perversions originate and to decide whether we are dealing with the atonic or the hypertonic form. The treatment of these conditions will vary according to this diagnosis.

Causes and Origin of Motor Insufficiency and Ectasy.—We speak of motor or mechanical insufficiency if the stomach is incapable of propelling the ingesta into the intestine within the normal time.

This definition indicates the path along which the diagnosis of such an insufficiency should travel. Essentially, the same methods can be employed that Leube[1] employed in his day for determining the time of digestion. If a test-meal is administered to a healthy subject and the stomach pumped out seven hours later, the organ will be found empty. In pathologic cases, on the other hand, a certain quantity of food will frequently be found at this time. The quantity of the residue will naturally vary in different cases. As a matter of fact, there are a number of cases on record in which the stomach was unable at any time to get rid of all its contents.

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[1] *Berichte der Rostocker Naturforscherversammlung*, 1871; *Deutsch. Arch. f. klin. Med.*, vol. xxxiii.

Leube, in these cases, speaks of a prolonged period of digestion; nowadays we designate these cases as motor or mechanical insufficiency or atony. Rosenbach[1] deserves the credit of having shown that the anatomic methods for measuring the size of a dilated stomach are insufficient, and that it is necessary to examine the functions of the organ in order to gain an insight into the motor powers of the organ. In the case of the heart, for instance, we are not able fully to determine the power of the organ by determining its size; more is needed, for we must analyze the tension, the amplitude, and other properties of the pulse. Rosenbach has called attention to the fact that it is impossible, in the same sense, to determine the powers of the stomach by measuring its boundaries, and that it is absolutely essential carefully to determine its functional powers. To continue Rosenbach's comparison, I might say that it is not immaterial to know whether or not the stomach is of normal size, even though we find out that its powers are reduced. The same, of course, applies to the heart. As in the case of the heart it is quite important to know the boundaries of the organ, so in stomach-diseases we must always attempt to determine whether or not there are excessive distention and dilatation.

Rosenbach has chosen the term "insufficiency of the stomach" for this condition, for the reason that we are dealing with a weakness of the stomach-wall and insufficiency of its propulsive powers.

Bouveret recommends the word "hypotonia" instead of the usual word "atony," which certainly does not completely designate the character of the functional perversion. He bases his recommendation on the fact that in these cases there is not a complete loss of tone, but merely a reduction in the normal tone of the stomach. Boas recommends the term myasthenia gastrica (from *μῦς*, muscle-fiber, and *ἀσθενία*, weakness). [Einhorn prefers to use the term ischiochymia rather than motor insufficiency of the stomach to designate this condition.—Ed.] I think the most appropriate designation is "motor or mechanical insufficiency of the stomach," for it applies to all forms, even to those in which the motor powers of the stomach are insufficient, though there is, at the same time, no direct loss of muscle power; in other words, to those cases in which the insufficiency of the organ is due to some obstacle to the propulsion of stomach-contents. The term atony should be applied only to those cases in which there is a direct reduction in muscle power or the muscle tone.

We can speak of ectasy only if this motor insufficiency is combined with a considerable enlargement of the organ. This abnormal distention may be transitory. As soon as a patient with motor insufficiency, particularly of the atonic kind, forces the stomach to an excessive amount of work, the organ may readily become dilated. If this excessive work is imposed on the stomach for a long period of time, we ultimately see a permanent abnormal distention that we call ectasy proper.

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[1] *Volkmann's Samml. klin. Vorträge*, 1878, No. 153.

In order that motor or mechanical insufficiency be produced, the normal relation between the elasticity and the muscle power of the organ, the propulsive forces of the organ, on the one hand, and the amount of work that is required of these forces, on the other, must be disturbed. In other words, the powers of the stomach must become incapable of performing the work that is demanded of them.

In all those cases in which the stomach is incompetent to propel its contents through the pylorus into the intestine or in which this function is imperfectly performed or not performed within the normal time, we are dealing with that perversion of function that we call mechanical or motor insufficiency. It may be due to the following factors: First, the quantity of material to be propelled may be abnormally large; second, the propulsive forces may be reduced; third, there may be abnormal resistance to the exit of ingesta at the point of exit—namely, at the pylorus or its vicinity. Strictly speaking, the latter class of cases,—they are particularly cases of stenosis of the pylorus,—whether they are caused by carcinomata, cicatricial contractions, adhesions, or other factors, should not be called insufficiency proper. If we include this form under the heading of insufficiency, and if we discuss it in this place, we do this because it is characterized, in common with the other forms, by a prolonged period of digestion, an insufficient or delayed propulsion of ingesta, and a tendency to dilatation.

The genesis of these different forms varies. In those instances where motor insufficiency is due to some obstacle in the pyloric region there is no atony nor hypotonicity, but only relative insufficiency, for the muscular powers of the stomach are not reduced. The cause of this condition is the abnormal resistance that is opposed to the exit of the ingesta.

In the first two groups this is different: in the one case we are dealing with a primary reduction or loss of muscle power; in the second, with direct impairment of this muscle power from overwork. The latter form is the more frequently seen. The ultimate result in both cases is the same, for in both the muscularis is directly damaged and weakened—that is, it becomes atonic.

The last-named perversions originate whenever too much work is imposed on the stomach, either for a short or a prolonged period of time; occasionally we see, therefore, atony develop acutely after some dietary indiscretion—for instance, after eating too fast or after overloading the stomach with indigestible food. As a matter of fact, these acute forms of atony may be accompanied by a transitory dilatation of the stomach. Unless the stomach-contents is examined, this condition may readily be overlooked or be taken for a simple case of acute gastroptosis. In children particularly we see acute atony and ectasy following indiscretions in diet. In adults, particularly in subjects who are reduced, it can also be occasionally seen. In some of the cases that have been reported, the course of the affliction was very violent; in some instances death resulted. Slight degrees of this acute atony are probably more frequent than we suspect.

While it is true that in these cases of acute insufficiency and dilatation indiscretions of diet are the direct cause, it seems difficult to understand why such very severe forms are rare, as great indiscretions are undoubtedly frequently committed.

Certain predisposing factors play a rôle in these states. In many of the instances the patients were in the habit of overeating; in other cases that are reported the subjects were anemic or greatly reduced by some other disease; in still other cases a certain number of gastric symptoms were present for some time before.

All these factors must certainly be considered as predisposing agents in the sense that they caused a certain weakness of the stomach so that great indiscretions in diet could more readily lead to relaxation and even acute dilatation of the stomach.

The more chronic form of atony is seen in persons who are in the habit of overloading the stomach with indigestible food; in gourmands, in people who eat very rapidly or who live on a vegetarian diet; persons who drink very much also often develop this kind of atony, although here it develops less frequently than in those who eat too much. A milk-cure occasionally produces this condition, for many subjects who undergo such a course of treatment think that they will be benefited more if they drink as much milk as possible. Excessive drinking of other beverages, as coffee, soup, and beer, particularly drinking beverages that contain a great deal of gas, all damage the tone of the stomach muscles.

Another cause of this condition, though a rare one, is permanent regurgitation. In these cases the bile probably disturbs digestion in the stomach, and in this way constitutes a predisposing factor for the development of atony.

In the second place we see atony in cases of primary weakness of the muscularis, even though no excessive work is thrown on the stomach. There are cases in which this muscular weakness appears to be congenital and where it is transmitted from parent to child. A so-called weak stomach is hereditary in certain families. In the majority of cases, however, the method of feeding the children can be made responsible. The stomach, it may be said, is not trained correctly during childhood and adolescence, and is forced to master a diet that is not suitably selected. As soon as an excessive amount of work is thrown on the stomach, symptoms of insufficiency appear; in other cases, again, relaxation of the stomach may only be one symptom of a general weakness that may be either congenital or acquired. Atony of this character is seen in subjects who are underfed or in subjects who are weakened by excesses of all kinds, particularly in the sexual sphere. Atony of this kind may also appear acutely after violent emotional disturbances. This need not surprise us, for we see symptoms of irritation or of paralysis in the different nervous areas following violent emotional excitement; no wonder, therefore, that under these conditions the appetite may be suddenly lost or there may be excessive peristalsis, or atony and paralysis of the stomach may develop.

Some authors claim that organic lesions of the central nervous system may lead to atony. This point, however, has not been established beyond cavil, although it is highly probable that some cases may originate in this way.

Other cases have been observed in which a blow in the region of the epigastrium led to atony; in other cases, again, trauma caused perigastritis, and motor insufficiency developed as a consequence.

Some claim to have seen the development of atony after traumata that did not affect the region of the stomach itself. Acute and subacute peritonitis, moreover, which, as we know, occasionally leads to paralysis of the intestinal muscularis, may also affect the muscularis of the stomach in the same way. Injuries to the abdominal region, particularly following surgical interference, may, for the same reason, occasionally lead to atony and acute ectasy. Koeberlé reports cases of acute dilatation of the stomach in peritonitis following ovariotomy; Gross and Thiebaut, following an operation for incarcerated hernia; Peter, in diaphragmatic pleurisy.

Epigastric hernias may also lead to motor insufficiency. Here it is probable that portions of the mesentery that are incarcerated in the hernial sac exercise traction on the stomach and in this way impede its movements.

Irrational clothing, particularly in women, has also been designated as a predisposing cause for atony; the same applies to sedentary habits, particularly in subjects that live high. Repeated pregnancy in rapid succession has also been considered a predisposing factor; it is probable, however, that the latter cause primarily leads to postural anomalies of the stomach, and only secondarily to atony and ectasy.

Atony is particularly frequent in diseases of the stomach itself—for instance, in acute and chronic gastritis, in nervous dyspepsia, in hypersecretion.

Ulcer of the stomach is only rarely complicated by atony; at least, that has been my experience. Many authors report that atony may be quite pronounced in this lesion; it is true that in cases where an ulcer leads to stenosis of the pylorus there may be motor insufficiency and the ingesta may be propelled with difficulty, but the ulcer *per se* rarely leads to atony.

Occasionally we see gastroptosis and other dislocations of the stomach together, combined with atony and ectasy, or there may be some inflammatory process that extends from the serous lining of the stomach to the muscularis and leads to fatty degeneration and atrophy, in this way causing atony and even ectasy. Eroding substances may damage the muscularis in the same way without causing any cicatricial contraction of the pylorus.

Some writers claim that narcotics, particularly alcohol, may relax the musculature of the stomach. Bouveret attributed such an effect to the excessive use of tobacco, and claims frequently to have seen motor insufficiency of the stomach in subjects who smoked excessively; I should say, however, that in those cases we are dealing with gastritis that secondarily involves the muscularis of the stomach.

Boas calls attention to the frequent occurrence of atony in cholelithiasis, and I can corroborate this observation. In other affections of the liver and in diseases of the heart and kidneys atony is occasionally seen in the later stages.

[Glyn[1] supports the view that dilatation of the stomach is sometimes idiopathic in its nature, and finds it, accordingly, more frequently in males than in females, and principally in youths, accompanied by neurasthenia and hereditary weakness. Associated with dilatation were found a variety of pathologic conditions, including disturbances of secretion, gastritis, vomiting, and constipation.—Ed.]

After severe infectious diseases, in chlorosis and anemia, atony is occasionally seen. Here atony is not due to these diseases direct, but to some accidental injury to the stomach, particularly to dietary indiscretions that injure the organ. In many cases, particularly in chlorosis, the development of atony may be favored by the dislocation of the abdominal viscera which is so frequently observed in this condition. Chlorosis and anemia *per se* rarely lead to atony.

These factors constitute only a few of the many possible causes that can lead to motor insufficiency. We have enumerated chiefly those that lead to a particular form of this disease, namely, the atonic form of insufficiency or atony. In another group of cases there is no real loss of tone of the muscles nor of their power, but, on the contrary, an increase; nevertheless, the propulsion of the ingesta is rendered more difficult. All processes that offer an abnormal resistance to the exit of the ingesta belong to this group. In many of these cases, of course, degeneration of the muscularis may develop secondarily as a result of the permanent overexertion of the muscles of the stomach-wall.

Stenoses of the pylorus or of the vicinity of the pylorus are representative lesions of this group.

There are rare cases in which the pylorus is originally narrow—so-called cases of congenital stenosis of the pylorus. In many instances this narrowing may be so great that children die a few days after birth; in other cases again the symptoms of motor insufficiency with dilatation of the stomach develop. Finkelstein and Grau[2] have recently described a number of cases of this character. As a rule, there was a considerable increase in the circular musculature of the pylorus. Stenosis of the pylorus and ectasy following reduplication of the mucous lining have also been observed.

[Examples of congenital pyloric stenosis continue to increase, and its etiology offers an interesting field for study. The disease seems to belong to a different group from the hypertrophic stenosis that develops later in life, which apparently results from long-continued spasm of the part or from inflammation in the submucosa. In the former group the cases appear in very young infants, in which there is sometimes vomiting from birth, and seems hardly to be accounted for on the theory of spasm or inflammation. It has been stated that the pyloric thickening is the result of an inflammatory process, but the histologic examination of some of these cases would seem to contradict this theory. Rolleston and Hayne[1] describe a case and collected 17 additional cases that had been reported by others. Henschel[2] reports 3 cases having occurred in the same family, which remarkable fact leads one to suspect that the affection is some form of congenital dystrophy. Some cases have been reported in which there was found an impervious pylorus. Thompson[3] reports 3 cases and subscribes to the belief that the condition is the result of a functional disorder of the nerves of the stomach and pylorus, with an "ill-coordinated, and therefore antagonistic, action of the muscular coat." Meltzer, in his studies of the affection,[4] believes that it is simply an embryologic malformation, a view that seems to accord best with the facts. The disease is to be recognized from the persistent vomiting, gastric dilatation, and inanition. Besides this, a perceptible induration is sometimes discoverable by palpation. Some cases have been greatly benefited by lavage, when the trouble has sufficiently receded to admit of moderate assimilation; but although the child may live, it is undersized and poorly developed. Various operations have been recommended, and some have proved successful. The first recorded favorable result was the case of Abel,[5] who performed a gastro-enterostomy on a male infant eight weeks old.—ED.]

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[1] *Boas' Arch.*, vol. vi., No. 1.

[2] *Jahrb. f. Kinderheilkunde*, vol. xliii.

Stenosis of the pylorus, in the majority of cases, is acquired. The most frequent causes of this lesion are cicatrices from ulcer, carcinoma of the pylorus, and cicatrices following erosion of the mucous lining of the stomach by irritating substances that have been swallowed. Narrowing of the lumen of the duodenum may lead to insufficiency and dilatation of the stomach in the same way as narrowing of the pylorus. The symptoms, of course, will vary according to the location of the obstruction, and will be different if the stenosis is above the papilla than if it is below; in the latter case the stomach will contain bile constituents, and a permanent regurgitation of bile will probably occur. In the former case this will not be seen. The permanent regurgitation of bile into the stomach aids the further development of the ectasy, for it interferes with digestion; so that in those cases where there is a mechanical obstruction in the duodenum,—a duodenal stenosis,—ectasy is produced, both by the lesion itself and by the action of the bile. Stenosis of the duodenum may be caused by ulcers, cicatrices following ulcers, carcinoma, compression of the duodenum from without, adhesions, dislocation of the parts by traction, knicks in the intestine, or even diverticula of the duodenum.

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[1] *Brit. Med. Jour.*, April 23, 1898.

[2] *Arch. f. Kinderheilkunde*, vol. xiii., p. 32.

[3] *Edinburgh Hosp. Reports*, vol. iv.

[4] *Medical Record*, July 20, 1898.

[5] *Münch. med. Wochenschr.*, November, 1898.

Acquired hypertrophy of the musculature of the pylorus is a less frequent cause of gastric insufficiency and ectasy; this condition is occasionally seen to follow severe gastritis; in some cases of hyperacidity and hypersecretion or in ulcer the repeated attacks of spasmodic closure of the pylorus may also ultimately lead to muscular hypertrophy. Simple spastic stenosis may also occasionally lead to motor insufficiency that, as a rule, however, is temporary. These spastic stenoses are seen in inflammatory and ulcerative processes involving the pyloric portion of the stomach; they are also occasionally seen in hyperacidity and hypersecretion.

Other rare causes of stenosis of the pylorus and secondary motor insufficiency are benign tumors of the pylorus, pedunculated polypi, connective-tissue hyperplasia of the pyloric region, occlusion of the pylorus by adhesions with neighboring organs, tumors that press upon the pylorus from without (large gall-stones in the gall-bladder—Minkowski), perigastric adhesions, adhesions with the gall-bladder, etc.

[Some time ago Boas, under the title of "Stenotic Hypertrophic Gastritis," described a condition resembling that which Habershon had previously characterized fibrous disease of the pylorus, and Tilger as stenotic hypertrophy of the pylorus. The term "linitis plastica" has likewise been employed to describe this affection, but this term is also used to designate a number of other stomach conditions of different character. The chief characteristics of the affection are a slowly progressive stenosis of the pylorus resulting in food stagnation, and often in a perceptible, but benign, tumor in the pyloric region. The chemistry in one case under my observation was, as stated by Boas, unlike that found in cancer, but pointed rather to a benign process. Boas recommends drainage by gastro-enterostomy, and has had successful results.

Oettinger[1] reports a case of extensive sclerosis in the pyloric extremity of the stomach causing stenosis, with the usual sequels. It was operated, much of the pylorus resected, and this resulted in recovery. Histologic examination showed the induration to depend upon an extensive fibroid degeneration of the submucous tissues depending upon inflammation. This condition if not precisely identical with, at least closely resembles, the stenosing gastritis described by Boas.[2]—Ed.]

The degree of motor insufficiency varies in individual cases. Sooner or later the typical picture of dilatation of the stomach develops in most of these cases. The most severe forms of dilatation are seen in stenosis of the pylorus, but even simple atony may occasionally lead to ectasy. This atonic form of ectasy is essentially different, however, from that form of ectasy that is caused by stenosis, as the latter is always complicated by hypertrophy of the musculature. The same applies here as in stenosis of one of the valves of the heart; here we see hypertrophy and dilatation of those portions of the heart that are situated behind the obstruction; in stenosis of the pylorus, in the same way, we see hypertrophy of that portion of the stomach that is situated immediately behind the obstruction—namely, the region of the pylorus. In the case of the heart, dilatation soon supervenes, and the same applies in the case of the stomach, for here ectasy gradually develops and slowly grows more severe. In the atonic form of ectasy, on the other hand, the muscularis remains very thin and atrophic.

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[1] *La Semaine méd.*, May 7, 1902.

[2] *Arch. f. Verdauungskrankheiten*, vol. iv., No. 1.

In many instances the morbid process that has caused the stenosis—for instance, carcinoma—finally involves the muscularis itself; when this occurs, compensation is rendered more difficult. Inversely, ulcerative disintegration of a carcinoma may lead to an improvement in the stenotic symptoms; this relief, however, is rarely sufficiently pronounced to cause a full compensation and a restitution to normal.

In conclusion, a few words may be permitted in regard to the much-discussed relations between floating kidney and dilatation of the stomach. Bartels[1] was the first to express the opinion that floating kidney of the right side, by exercising pressure on the duodenum, quite frequently leads to gastrectasy. Other investigators have advanced the opposite opinion—namely, that ectasy is the primary feature and floating kidney a secondary result of this condition; they imagine that floating kidney is produced by pressure of the dilated stomach on the abdominal organs and on the kidney itself. Still others—and they constitute the majority—deny any causal relation between floating kidney and ectasy. It is true that in many cases of floating kidney the greater curvature is lower down than normal, but this is not the result of ectasy, but of gastroptosis. Both conditions—floating kidney and gastroptosis—are independent of each other and both are the result of a third common cause. Formerly a low position of the larger curvature was interpreted to signify ectasy (even to-day this mistake is made), but in cases of this kind we are frequently not dealing with ectasy, but simply with a stomach that is dislocated downward. We have already mentioned that gastroptosis may secondarily lead to ectasy. Gastroptosis and floating kidney are particularly common in women. We refer to the section on Gastroptosis and Enteroptosis for the details of this subject.

[**Post-operative Dilatation.**—Fenger, Box, and Wallace, Kundrat, Schnitzler, Albrecht, and P. Muller have all contributed to the subject. The last-named observer reports 5 cases and throws much light on the pathology. From the present standpoint, it seems clear that most of the cases of post-operative dilatation so far reported are secondary to occlusion of the duodenum or some other portion of the intestine, resulting from pressure by cicatricial bands, or some other event connected with the operation or with the preceding disease. At the same time, it should not be overlooked that certain cases result from trauma, as, for example, that reported by Appel,[2] in which a fatal dilatation resulted in a boy who two weeks previously sustained a fracture of the left femur, caused by the caving upon him of a bank of sand. The patient was doing well a week following the injury, but soon began to suffer from dilatation. An exploratory examination was made, but no evidence of obstruction could be discovered. From this and from some other operative cases carefully examined postmortem it would appear that the dilatation occasionally developed without any obstruction of the intestine having been demonstrated. Cohnheim[1] concludes that as a result of trauma there may develop a chronic dilatation of the stomach, but that this is usually owing to the setting-up of some obstructive process at the pylorus or below. The practical inference may be drawn from our present knowledge that in all cases following operation in which vomiting is persistent, careful examination should be made as to the possible existence of acute dilatation of the stomach. When it is recognized, gastric lavage should be practised for the relief of vomiting, and if the vomiting recurs, the surgical indications are plain.—Ed.]

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[1] "On Abnormal Motility of the Right Kidney and its Connection with Dilatation of the Stomach," by Müller-Warneke, *Berlin. klin. Wochenschr.*, 1877, No. 30.

[2] *Phila. Med. Jour.*, August 12, 1899.

Pathologic Anatomy.—Motor insufficiency, both of the atonic and the hypertonic form, shows no anatomic lesions provided the case comes to autopsy before the condition is very far advanced; this perversion of function, therefore, can be recognized only by clinical observation. Dilatation proper, however, has a pathologic-anatomic basis. The pathologic anatomist will only in rare instances be able to say that the motor power of the stomach was disturbed during life, particularly in those cases where the stomach is not found dilated. Only in rare cases of chronic interstitial inflammation of the stomach, in cirrhosis of the stomach, and in diffuse forms of carcinoma do the anatomic findings justify us in concluding that there was motor insufficiency during life. We refer to the sections on these different diseases for the details; in this place we will discuss only the anatomic changes caused by dilatation and hypertrophy of the gastric muscularis.

The stomach may be either partially enlarged or enlarged in its totality. Partial dilatation in rare cases assumes the form of diverticula. These are usually caused by prolonged pressure on a circumscribed portion of the stomach, either by solid bodies that are contained within the organ (coins, etc.), or by adhesions that exercise traction from without.

General dilatation of the stomach is more important and more frequent. This condition may be seen in different stages. Dilatation usually begins at the lowest portion of the stomach,—that is, the fundus and the greater curvature,—because these parts of the organ have to sustain the greatest weight. In cases where the stomach is dislocated and is seen in an abnormal position, the dilatation is often found in the most dependent portion of the organ, and the position of the stomach will usually determine whether the pyloric region or the infundibulum is involved.

Sooner or later general dilatation develops. The degree of enlargement may vary greatly; there are cases on record in which the stomach reached as far as the symphysis and occupied nearly the whole left half of the abdomen. The most severe degrees of dilatation are seen in stenosis of the pylorus.

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[1] *Arch. f. Verdauungskrankheiten*, vol. v., p. 405.

The walls of the stomach present a different appearance in different cases. They may be either thick or thin. The condition of the walls of the stomach depends, on the one hand, on the primary disease that is causing the ectasy; on the other, on the duration of the affection.

Severe degrees of hypertrophy involving particularly the pyloric portion of the stomach are seen chiefly in cases in which stenosis of the pylorus or of the pyloric region caused the ectasy. In cases of this character the musculature of the pyloric portion of the stomach may be three or four times as thick as normal. The muscularis of the fundus of the stomach is, as a rule, not thickened; it may, in fact, be very much thinner than normal; in other cases hypertrophy of the pyloric musculature is not seen, but the whole gastric musculature is uniformly thin and atrophic. The latter condition is found particularly in cases of so-called atonic ectasy; in cases of very severe ectasy there may even be diastasis of the musculature.

Microscopic investigation reveals different findings: in the earlier stages there are no microscopic changes in the musculature. In ectasy of long standing atrophy and fatty degeneration of muscle-fibers are seen. Kussmaul and Meyer have also reported the occurrence of colloid degeneration.

The appearance of the mucosa and submucosa will naturally vary according to the primary cause of the disease. In carcinoma of the pylorus the well-known features of atrophic gastritis in its different stages are discovered. In some cases the signs of an ordinary gastritis are seen. In still others the primary cause that produced ectasy and atony also produced gastritis; or, again, there may be secondary inflammatory irritation of the mucosa following the stagnation of the ingesta. No typical picture of this condition can be delineated, owing to the variety of causes that may lead to this form of ectasy.

When the stomach becomes dilated it is usually distended in the direction of least resistance; consequently different abdominal organs may be dislocated by the stomach. The small intestine is usually pushed out of place—as a rule, downward, less frequently upward; the transverse colon may become dislocated; the liver and the spleen pushed upward. There are, however, a number of cases on record in which the enlargement of the stomach occurred upward; in one interesting instance the lung on one side was contracted so that the diaphragm stood particularly high; the stomach in this case was dilated at the same time, and the enlargement occurred upward into the convexity of the diaphragm.

Rosenstein[1] has recently reported a case that is unique; in this patient the stomach was enlarged upward and to the left, so that a disease-picture was simulated that resembled left-sided pneumothorax.

Symptomatology.—In studying the symptomatology of gastric insufficiency and ectasy those symptoms that are due to these states *per se* must be distinguished from those that are caused by the primary disease that leads to these perversions or by other lesions or perversions that may exist at the same time. Cases of motor insufficiency occur that produce no symptoms whatever; others again produce very severe and quite typical symptoms.

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[1] *Arch. f. d. Verdauungskrankh.*, vol. ii.

It will, of course, be quite impossible to discuss the symptomatology of all those diseases that may lead to insufficiency and ectasy of the stomach; we are forced to limit ourselves to a description of those symptoms that are characteristic for these perversions. At the bedside we proceed step by step in our diagnosis and determine first the existence of ectasy and later that of motor insufficiency. Then we attempt to determine the character of the ectasy and its origin; we also attempt to decide whether we are dealing with the atonic form of ectasy or with an ectasy that is the result of stenosis near the pylorus.

If we have arrived at the conclusion that the latter form of ectasy is present, it remains to determine whether the disease is malignant or not. The symptom-complex of motor insufficiency and of ectasy is essentially the same in all cases; what differences there are are differences of degree and not of kind. The symptom-complex will, however, vary according to the primary cause of the disease, and a different syndrome will be presented in cases of ectasy and motor insufficiency that are due to carcinoma of the pylorus leading to stenosis and obstruction, to an ulcerative cicatrix causing obstruction of the pyloric region, or to atonic ectasy, etc. The most important symptoms, therefore, are those that are caused by motor insufficiency; after these have been studied we must investigate the symptoms caused by the ectasy of the stomach. These are usually identical with those caused by motor insufficiency, with this distinction, however, that wherever there is ectasy, the stomach is abnormally enlarged.

In the introduction I called attention to the fact that ectasy and motor insufficiency of high degree frequently occur together, but that they are not necessarily combined in all cases. In practice it is a good plan to regard motor insufficiency as a simple perversion of function and ectasy as a perversion of function that may lead to anatomic changes; in other words, it is wise to determine whether or not the insufficiency has led to permanent enlargement of the stomach.

One form may gradually merge into the other, so that it may be doubtful in many cases whether we are dealing with motor insufficiency alone or whether slight degrees of ectasy exist. In pronounced cases this decision is not difficult to render.

In practice it will be found that severe forms of motor insufficiency that persist for a long time frequently lead to abnormal distention of the stomach and to ectasy; we may, therefore, say in general that the prognosis of those forms of motor insufficiency that persist for a long time and still do not lead to ectasy is comparatively favorable.

In this place we must discuss primarily those symptoms that indicate that the power of the stomach to propel its contents into the intestine within the normal time is insufficient. I feel justified, therefore, in discussing those symptoms first that indicate the existence of this perversion directly. Later I will discuss the objective symptoms, particularly those that reveal the existence of an abnormal enlargement of the stomach; and, finally, I will discuss the subjective symptoms of these lesions and perversions.

The only way in which to demonstrate in any given case that the motor power of the stomach is insufficient is to make a direct examination of this function. There are different ways of doing this: despite all the efforts that have recently been put forward to devise improved methods, the old procedure of testing the time of digestion, as advised by Leube, is still the best and the most reliable method we possess to-day.

In a healthy subject the stomach will be found to be empty six to seven hours after the ingestion of the test-meal that I have recommended. If the stomach is pumped out at this time or later, and if a considerable quantity of undigested food is found, the stomach may be called insufficient. Whether this is a genuine primary form or a secondary form is not determined by this test. In order to render a decision in this direction further examinations must be carried out that are directed particularly to an investigation of the gastric secretion and similar factors.

The degree of motor insufficiency varies greatly in individual cases. A general estimate of the degree of insufficiency may occasionally be gained from an examination of the quantity of residue found in the stomach at a certain time after the ingestion of a test-meal. If, for instance, 300 c.c. of food residue are found in a given case seven hours after the ingestion of a test-meal, and if, in another case in which the same amount of food is eaten, 600 c.c. or more are found at the same time, the latter case will be more insufficient than the former one.

A great number of variations and degrees can naturally be distinguished, and each physician will have to decide for himself how many subdivisions he wishes to arrange. The majority of clinicians distinguish two degrees of motor insufficiency: if remnants of food are found in the stomach in the morning after a simple supper, this is usually considered a severe degree of motor insufficiency; if, on the other hand, the stomach contains no food in the morning, but does contain some remnants of food seven hours after a test-meal, then we usually speak of a mild degree of motor insufficiency.

The method of examination that we are discussing can naturally be modified in many directions. I usually proceed in a slightly different way from other clinicians. I administer a test-meal and wash out the stomach some six to seven hours afterward; if remnants of food are found at this time, the proof is given that the patient is afflicted with motor insufficiency. The degree of insufficiency is also approximately indicated by the amount of residue; if the stomach is very much dilated and if an abundant quantity of residue is found when the stomach is washed out late in the afternoon, I usually wash the stomach out thoroughly and then instruct the patient to eat a simple supper. The next morning before breakfast the stomach-contents is again pumped out; if there is only a mild degree of motor insufficiency, the stomach will be found empty. This proves that the stomach is incapable of getting rid of its contents seven hours after a test-meal, but is capable of getting rid of a small meal overnight—that is, during a longer period of time. There are still other cases in which a large or a small amount of residue is found early in the morning before breakfast. It is clear that a case of the latter kind is afflicted with a higher degree of motor insufficiency than any of the above.

I also sometimes proceed as follows: On one day I wash out the stomach in the evening before supper, then I administer the supper and wash out the stomach-contents the next morning. On the second day I do not pump out the stomach before supper, but again remove the stomach-contents the next morning before breakfast. In one series of cases it will be found that the stomach contains a considerable amount of food residue in the morning—on those days, namely, on which the stomach was not pumped out the evening before; in the other series it will be seen that the stomach is empty the next morning, provided the stomach was washed out the evening before. This experiment demonstrates that washing out the stomach in the evening has certain advantages. I have a patient in my clinic to-day who is afflicted with ectasy, stenosis of the pylorus, and hyperacidity, and in whom 450 c.c. of residue were found in the morning. If the stomach was washed out the evening before, nothing was found in the morning. By washing out the stomach before supper we accomplish, this, therefore, that the stomach is empty in the morning; in other words, that the organ is less taxed during the night than if the organ had not been washed out in the evening. The main indication in the treatment of these cases is to avoid overtaxing the stomach, and in this instance lavage of the stomach in the evening certainly accomplished this object better than lavage in the morning.

It is well to administer the same test-supper in those cases in which it is desired to draw diagnostic conclusions from the presence or absence of food-remnants in the morning. Boas has recommended a test-supper consisting of cold meat, white bread, butter, and a large cup of tea.

The test-supper can, of course, be arranged in many different ways. I am of the opinion that in those cases where a large amount of residue is still present in the evening the stomach should first be washed out, the supper then administered, and the stomach washed out again the next morning before breakfast. If aspiration of the stomach-contents is performed only in the morning and if a large amount of residue is found at this time, we do not know how much of the food eaten at dinner-time was present in the stomach when the supper was administered. If the test, therefore, is performed in this way, we are hardly ever justified in stating that the stomach was unable to master the supper during the night, for we know that the stomach was not empty at the time when the supper was eaten.

It is not surprising that severe and extreme degrees of motor insufficiency, as a rule, lead to enlargement of the stomach and to ectasy, and that in those cases where the stomach is permanently overburdened, the organ gradually enlarges.

The salol test of Ewald and Sievers is another good method of testing the motor powers of the stomach. In regard to the details of this method, its applicability and its value, I refer to what has been said about it on page 138. The most reliable modification of this salol method is the one suggested by Huber. This author does not determine the time at which the salicyluric acid reaction appears, but estimates the motor powers of the stomach from the length of time during which the reaction can be elicited. In a healthy subject the salol reaction (violet coloration of the urine on addition of a dilute solution of chlorid of iron) disappears after twenty-six to twenty-seven hours; if it persists for a longer time, this indicates motor insufficiency. The method is not, however, a positive criterion for the existence of this condition.

The oil method of Klemperer and the modification suggested by Mathieu and Hallot is not well adapted for practice. The most practical method is unquestionably the one described first—namely, the aspiration of the stomach-contents after a certain stated period of time after a test-meal.

The succussion sound is of some value in determining the existence of dilatation of the stomach. It is employed in the same way here as in the diagnosis of mechanical insufficiency. It is true that the succussion sound itself has no diagnostic value; I cannot emphasize this sufficiently; it is important and significant only if the time is considered at which it can be elicited and the exact location in which it can be heard. It is altogether wrong to consider the presence of succussion sounds a proof of atony or ectasy; this is frequently done in practice, but is radically wrong.

If a healthy subject is examined two hours after dinner, particularly if he has swallowed much fluid, or half an hour after drinking two cups of tea, a distinct succussion sound can usually be elicited. This is, of course, not surprising, for the succussion sound simply indicates that fluid and air are present in the stomach at the same time. If, on the other hand, succussion sounds can be heard seven hours after the ingestion of a test-meal or two hours after a test-breakfast, or in the morning before breakfast, this demonstrates that the stomach is not empty at a time when it should normally be empty; in other words, that there is motor insufficiency. The important feature, therefore, in utilizing the succussion sound for diagnosis is not to determine the mere occurrence of this phenomenon *per se*, but to determine the exact period at which it can be heard.

The second important factor is the determination of the exact location in which the sound can be heard and the extent of the whole area over which it can be elicited. This test does not teach us whether the stomach is insufficient, but merely indicates whether the stomach is dilated or not. I mention these points in this place because I wish to advise all those who perform the succussion test to determine not merely the appearance of the sound, but also its location and the area over which it can be elicited.

If succussion sounds can be heard seven hours after a test-meal far below the umbilicus, this shows that the lower boundary of the stomach is situated further down in the abdomen than normal, provided, of course, that the succussion sound is really produced in the stomach and not in the colon.

The fact that these sounds can be elicited below the umbilicus does not demonstrate the existence of ectasy unless it can at the same time be positively determined that the upper boundary of the stomach is situated in its normal position; in other words, unless it can be shown that the whole organ is not dislocated downward or situated in a vertical position. If the stomach is merely dislocated downward or if there is general gastroptosis, the succussion sound will naturally be heard far down in the abdomen, without, at the same time, indicating the existence of ectasy. The only way to avoid this error is to inflate the stomach with air or with carbonic acid gas.

In examining a case of this character we will, of course, not limit ourselves to those methods of examination that I have described. As in any other case, the examination of the stomach should be carried out with thoroughness in all directions. In the first place, the region of the stomach must be carefully inspected. It must be determined whether or not there is a protrusion; whether or not peristaltic movements are visible; the stomach must be palpated, and it must be determined whether its boundaries can be exactly delineated by palpation.

In all cases where the existence of ectasy is suspected an experienced physician will perform his examination of the stomach at a time when the results obtained will allow him to draw definite conclusions; in other words, he will prescribe a test-meal and examine the patient seven hours afterward. If it is found that the stomach is abnormally filled at this time, and if a distinct succussion sound can be elicited, the stomach-contents must be removed and carefully examined, and, if necessary, the exact boundaries and the position of the stomach be determined by artificial inflation of the organ.

Following this discussion of the methods that I employ for determining the existence of motor insufficiency directly, I will briefly describe a few of the objective symptoms that are observed in this condition. The most practical method of describing these symptoms will be to describe the course of an ordinary examination and to explain what can be discovered by the different procedures that are ordinarily employed. I will begin with inspection. In simple motor insufficiency this method of examination reveals nothing; in cases, however, in which the stomach is greatly distended, in which ectasy exists, the outlines of the enlarged organ can frequently be seen with the naked eye. The most conspicuous portions of the stomach are the greater curvature and that portion of the organ that is situated immediately above it. The lesser curvature cannot be seen if the stomach is in its normal position; in cases, however, where the organ is low down in the abdomen, as in gastroptosis or in vertical position of the organ, the lesser curvature can occasionally be seen. In cases of severe ectasy a considerable degree of gastroptosis is frequently found. If the region of the stomach is stroked or if we wait long enough, peristaltic movements may occasionally be seen within the enlarged boundaries of the stomach. They generally extend from left to right toward the pylorus. If these wave-like movements are not carefully studied, the impression may be created that they do not proceed in a regular manner, but run from right to left or from left to right; careful inspection, however, will reveal that these waves in general proceed in a regular manner in one direction. Such active movements of the stomach-wall seem to argue against motor insufficiency, for these movements seem to indicate that the stomach is working with greater energy than normal. If in a case of this kind we find that the stomach-contents is not propelled onward within the normal time-limit, or if we find that some food-particles always remain behind, this indicates that we are not dealing with a case of true atony, but that the motor powers of the stomach are only relatively insufficient; in other words, that the task imposed on the stomach is so great that, despite the increase in its motor powers, it is incapable of mastering it. Peristaltic waves and incomplete propulsion of stomach-contents will naturally suggest the existence of some obstacle in the region of the pylorus—as, for instance, a stenosis of the pylorus that prevents the exit of the ingesta from the stomach into the intestine.

I will not enter into a discussion of transillumination of the stomach in this place. This method is not universally employed because it yields no information that could not be obtained in a more simple manner.

Palpation is more important. By this method we cannot determine the existence of insufficiency, but we can discover whether or not the stomach is abnormally large. With a little practice it is usually easy to determine the boundaries of the stomach from the uniform tension and the peculiar resistance that the organ offers. Sometimes it is possible to palpate the end of the stomach-tube through the abdominal walls; if this is possible, the lower boundary of the stomach, or rather the location of the greater curvature, can be determined.

Percussion is also useful; here, too, we determine the existence of ectasy and not of insufficiency. Percussion may be of value, however, in the diagnosis of the latter condition by showing that the stomach is not empty at a time when it normally should be empty. Percussion, above all, enables us to determine the boundaries of the stomach, and in this way to decide whether or not the organ is enlarged. In cases where the stomach still contains fluid material, as in ectasy, it is well to examine the patient both in the erect position and when he is lying down. If percussion is performed with the patient in the erect position, beginning at the costal margin at the left side and percussing downward along the parasternal line or external to it, an area of dulness will be found at the height of the umbilicus or still further down if ectasy is present; as soon as the patient lies down this area of dulness disappears. The diagnosis is rendered positive if this dulness disappears after evacuation of the stomach-contents and reappears as soon as fluid is introduced into the stomach. If such an area of dulness is found at the height of the umbilicus or below this level, the diagnosis of ectasy can be made, provided the upper boundary of the stomach is found to be in its normal position. Even in ectasy, however, this lower boundary may be seen in different parts of the abdomen according to the amount of material that the stomach contains; this, of course, applies also to cases of simple atony, for here too the amount of material contained in the stomach frequently determines the position of the lower boundary of the organ.

We refer to what has been said above in regard to splashing. It is usually an easy matter to distinguish splashing produced in the stomach from similar sounds produced in the intestine; in case of doubt the colon should be inflated. If splashing is heard in the colon, thin stools should be evacuated; in cases of ectasy, however, we usually find the opposite—namely, constipation and very hard stools.

The method of Penzoldt-Dehio is also suitable for determining the lower boundary of the stomach and the tone of the organ. If a patient with motor insufficiency drinks two or three glasses of water in succession early in the morning before breakfast,—that is, at a time when the stomach should be empty,—and if then the extent of the stomach dulness is determined after each glass with the patient in the erect position, it will be found that in cases of atony the stomach dulness extends further down than normal. Penzoldt advises controlling this finding by subsequently removing the water from the stomach and then percussing again.

Artificial inflation of the stomach with air or with carbonic-acid gas is essential in making a diagnosis of ectasy, for the reason chiefly that this procedure gives us the most positive information in regard to the position of the upper boundary of the stomach—of the lesser curvature. For determining the existence of motor insufficiency the method is, of course, without value.

Inflation gives us information in regard to the size, the distention, the form, and the position of the stomach. In case the greater curvature is found low down, inflation may teach us whether the organ is abnormally large or whether the whole organ is situated low down in the abdomen—in other words, whether we are dealing with an enlargement of the stomach, with gastroptosis, or with vertical dislocation. In combination, therefore, with other findings this method is a valuable diagnostic adjuvant.

Chemical examination of the stomach-contents in motor insufficiency and ectasy naturally furnishes very different results. Everything will depend on the primary disease that causes the atony and stenosis.

We are rarely able to diagnose the primary disease from an analysis of the gastric secretion, but we can usually obtain some very valuable clues.

If, for instance, in a case of stenosis of the pylorus we find no free hydrochloric acid, but a large quantity of lactic acid, we can suspect a malignant form of stenosis and carcinoma; if, on the other hand, we find an abundant quantity of gastric juice that separates into three layers and gives strong hydrochloric acid reactions, we can infer that the stenosis is benign.

A special diagnosis of any one disease cannot, of course, be made from the chemical findings alone, but all other factors must be taken into consideration.

It may also occur that stagnated stomach-contents irritates the gastric mucous membrane, and in this way secondarily influences the secretion of gastric juice.

In advanced cases of insufficiency and ectasy fermentative processes are frequently encountered; there may even be gaseous fermentation. Numerous micro-organisms are constantly found in an ectatic stomach. If there is acid fermentation, there will be pyrosis and acid belching; if there is gaseous fermentation, the stomach will be inflated and there will be frequent belching; the stomach-contents and the vomit will always contain a large quantity of foam if there is abundant fermentation.

In order that fermentative processes may occur in the stomach the stomach-contents must stagnate. The character of the fermentation, however, is not at all dependent on the character of the gastric secretion. Lactic-acid fermentation is seen particularly in cases of carcinoma in which there is a deficiency of free hydrochloric acid; in other cases we see yeast fermentation and abundant development of gas. Cases are on record in which these gases were inflammable. Particularly those cases of ectasy seem to furnish a suitable medium for gaseous fermentation in which the values for hydrochloric acid are normal or increased. Occasionally gaseous fermentation is seen together with lactic acid fermentation, even though the stomach-contents is not acid.

Gaseous fermentation is never seen if the motor powers of the stomach are normal. For this reason the appearance of this symptom may be regarded as a positive sign of motor insufficiency or, better, of abnormal stagnation of stomach-contents. If the fermentation test is performed in the manner described in the general part of this volume,—namely, after the addition of grape-sugar,—a general estimate of the degree of insufficiency may be made from the rapidity with which fermentation occurs and the intensity of the process.

The exact analysis of the gases formed is of purely scientific interest, but of no value from a practical point of view. Combustible gases are occasionally developed; if these are gathered in a suitable vessel and then passed through a glass tube, they will burn with a bluish or yellowish flame.

Boas[1] has recently called attention to the occasional occurrence of sulphureted hydrogen in the stomach of cases of ectasy. He attributes the development of this gas to the putrefaction of albumin, and claims that H_2S is very frequently seen in cases of benign ectasy of the stomach. In carcinomatous cases he believes that it is constantly absent.

[1] *Deutsch. med. Wochenschr.*, 1892, No. 49; *Centralbl. f. innere Med.*, 1895, No. 3.

Sticker[1] recently performed a number of experiments in this direction in my laboratory and arrived at different conclusions. He found that in the majority of cases H_2S is not formed from the decomposition of proteid material, but that it is formed by the diastatic action of the saliva from certain vegetable oils that contain sulphur. According to these results, the appearance of H_2S would be of value only in the diagnosis of putrefactive processes in the stomach, if it can be shown that vegetables containing sulphur were not eaten before and that the stomach contains other products of putrefaction besides H_2S.

In some cases the stomach always contains bile. This is seen particularly in cases of ectasy that follow stenosis of the duodenum. Constrictions of the duodenum that are situated above the papilla lead to the same results as stenosis of the pylorus; constrictions, on the other hand, situated beneath the papilla, are distinguished from the former by the presence of bile in the stomach-contents. Several years ago I[2] reported a case of this character in which the duodenum was narrowed near the entrance of the bile-duct. The obdurator in this case was a large gall-stone that pulled the duodenum upward; the stomach, on the other hand, was pulled downward by abnormal adhesions that had formed particularly in the region of the pylorus; as a result of all this distortion the bile that was poured out of the common duct was forced backward into the open pylorus. This case terminated fatally. Before death the peptic power of the stomach was completely lost. Honigmann[3] reported similar findings in my clinic in a case of chronic narrowing of the intestine; here, too, the power of the gastric juice was almost completely lost on account of the constant regurgitation of bile into the stomach. As soon as the intestine became patent, so that none of the duodenal contents was forced backward into the stomach, gastric digestion again became normal.

Every case of stenosis of the duodenum does not naturally lead to such severe symptoms as those observed in the two cases described; much will depend on the amount of bile that regurgitates into the stomach, and a great deal will also depend on the character of the gastric secretion. Small admixtures of bile do not necessarily interfere with digestion.

There are finally cases in which pure gastric juice is found in the stomach instead of a mass of food. Such patients are suffering from hypersecretion, a secretory anomaly that is frequently combined with motor insufficiency and ectasy.

Microscopic examination of the stomach-contents reveals a variety of things in cases of motor insufficiency. In those cases, for instance, that are caused by carcinoma of the pylorus, numerous well-preserved meat-fibers are found. In cases in which the secretion of gastric juice is increased, there are abundant quantities of undigested starch; in carcinomatous ectasy the well-known long bacilli, are seen, whereas in the benign form numerous yeast-cells and sarcinæ predominate. I wish, however, to emphasize particularly that none of these findings are constant. The secretion of hydrochloric acid, for instance, may be stimulated by adequate treatment to such an extent that the different fermentative processes are altered.

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[1] *Münch. med. Wochenschr.*, 1896.

[2] *Zeitschr. f. klin. Med.*, vol. xi.

[3] *Berlin. klin. Wochenschr.*, 1887.

Absorption naturally suffers in advanced degrees of motor insufficiency. The iodin method is usually a good index of the degree of insufficiency. If there are subacidity and anacidity of the gastric juice, the digestion of proteids, and consequently their absorption, suffers. This does not necessarily imply that an abnormal proportion of albumin is wasted in the feces, for the intestine may vicariously assume the functions of the stomach; this can occur only, however, if the stomach-contents is emptied into the intestine within normal time-limits; if this does not occur,—as, for instance, in ectasy or atony,—abnormal fermentation and decomposition of the stomach-contents develop and absorption is consequently seriously interfered with.

The functions of the intestine are frequently involved in this disease. This need not surprise us, in view of the fermentative and putrefactive processes that occur in the stomach. In the atonic forms of motor insufficiency we consequently sometimes see atony of the intestine.

The stools in cases of advanced ectasy are usually sluggish—there is a tendency to constipation, less frequently to diarrhea. The fermentative and putrefactive processes that occur in the stomach frequently lead to analogous processes in the intestine, with the development of much gas; as a result, inflation of the intestine and atony of the intestinal cells may develop. The development of this condition is also favored by the small amount of water that passes through the stomach into the intestine; this leads to a loss of water in the tissues, so that more water is abstracted from the intestinal contents than normal; this also explains the hardness and dryness of the stools in these cases. The appearance of a normal stool is, therefore, a good prognostic sign in cases of ectasy that ordinarily are afflicted with obstinate constipation. Occasionally the intestinal walls are irritated by fermented and putrefied material that is poured into the intestine from the stomach; when this occurs, diarrhea may naturally supervene for a time.

The examination of the urine reveals some very significant features. The quantity is important; it may be normal in these cases, but is usually decreased in proportion to the degree of insufficiency and ectasy existing. It is not rare to find a daily excretion of 500 or 800 c.c. or less, even though the patient drinks abundant quantities of water.

The smaller the amount of water excreted, the more concentrated naturally the urine and the higher its specific gravity. On standing, a white sediment of phosphates, particularly of triple phosphates, frequently forms.

The reaction of the urine in cases of ectasy is frequently alkaline, particularly where there is an excessive production of hydrochloric acid. If much gastric juice is removed from the body by profuse vomiting or by frequent lavage, the urine may become neutral or even alkaline.

In cases of inacidity of the gastric juice the urine always remains acid, even though the stomach is frequently washed out and there is frequent vomiting.

Rare urinary findings that have nothing to do with the primary disease are the occasional occurrence of small quantities of albumin, of acetone, and of diacetic acid; the chlorids are usually reduced in advanced cases of ectasy. [An excess of indican is usually present in cases of intestinal fermentation.—Ed.]

The heart may also be involved. In severe cases of gastric ectasy the pulse may be considerably slowed; how this retardation of the pulse occurs is undecided; the fact, however, remains that in advanced cases of ectasy the pulse-rate may be reduced to 50 and even 40 beats in the minute. This phenomenon cannot be explained on purely mechanical grounds; some authors have attempted to explain it by the elevation of the diaphragm, but the fact that a similar retardation of the pulse-beat is found in cases in which the enlarged stomach does not force the diaphragm upward contradicts the assumption. As a matter of fact, retardation of the pulse-beat is occasionally seen in cases where the diaphragm is lower than normal.

Mild degrees of dyspnea are occasionally encountered in ectasy; this condition is rarely caused by the upward dislocation of the diaphragm and pressure on the heart and lungs, although many authors favor this explanation. To judge from my personal experience, elevation of the diaphragm rarely results from dilatation of the stomach. In ectasy the contrary is frequently seen—namely, a comparative lowering of the diaphragm and an enlargement of the boundaries of the lungs. It would lead us too far were we to discuss the origin of dyspnea exhaustively. Aside from the mechanical causes, certain nervous influences may produce this condition—as, for instance, irritation of the vagus. All we can say is that in the majority of cases this dyspnea cannot be explained from compression of the lungs by the ectatic stomach.

Some authors speak of asthmatic disturbances as cases of dyspeptic asthma. I do not favor this term, for asthma is a strictly defined clinical symptom-complex that is in no wise related to the disease that we are discussing. Asthma and dyspnea must be sharply distinguished. The occasional occurrence of dyspneic symptoms in ectasy, even if they occur in attacks, does not justify us in speaking of asthma.

The general health of the patient and the general nutrition do not suffer in mild degrees of motor insufficiency; in the more advanced forms, particularly those that are combined with ectasy, the patient may be very much reduced. The more advanced the ectasy and the longer its duration, the more does nutrition suffer. In cases where the secretion of gastric juice is greatly diminished, as, for instance, in carcinoma of the pylorus, nutrition is more impaired than in cases where the secretion of gastric juice is normal or even increased. Even in the latter class of cases, however, the state of nutrition ultimately suffers. One of the most conspicuous symptoms of advanced cases is the dryness of the skin and mucous membrane that results from the withdrawal of water from the tissues.

Finally, extreme degrees of emaciation may be seen; the adipose layer disappears completely, the patients appear weak and debilitated, the skin feels rough and dry and desquamates easily. The patients present a picture of general decay. Even in this advanced stage of the disease, and even though the ectasy of the stomach be far advanced, we may succeed in greatly ameliorating the condition of the patient by appropriate treatment, particularly by the introduction of water by the rectum and similar measures. This, of course, applies only to those forms that are not malignant.

In the preceding paragraphs I have briefly discussed the most important objective symptoms of motor insufficiency and ectasy. All other symptoms may vary greatly. In some instances insufficiency exists with slight objective symptoms or without any symptoms at all; in others again the disease-picture is very severe. There is really no characteristic symptom-complex for insufficiency and ectasy unless we consider the few direct signs that we have mentioned above as typical.

If we make it a rule to examine the motor power of the stomach in every case in which we test the secretory functions, we will be surprised to find slight degrees of atony in a great many cases; this atony may remain altogether latent; in other cases atonic states may appear for a short time whenever the stomach is overtaxed.

A patient with motor insufficiency of slight degree usually complains of a feeling of tension, pressure, and fulness in the stomach region after large meals; as soon as the stomach is empty these sensations disappear. The appetite at the same time remains good, and pain is not present. Gradually this feeling of pressure increases in severity, and whereas in the beginning of the disease it occurs only after excessive eating, it occurs even after small meals as soon as the disease is somewhat advanced; at this stage it also lasts longer. Cases of this kind experience a disagreeable feeling of fulness and discomfort immediately after eating; even the pressure of the clothing bothers them; in addition there is belching; the gas may either be tasteless or have the flavor of the food. These patients also frequently complain of heartburn, of acid or putrid belching, appearing immediately after eating, or in other cases later; in some cases it may persist beyond the normal time of digestion.

In other cases there is a feeling of unrest, of spasmodic to-and-fro movements in the stomach, immediately after eating. This is found particularly in cases in which there is some obstruction in the region of the pylorus. In these cases, too, we may frequently see peristaltic movements of the stomach if the abdomen is carefully inspected. The latter phenomenon can, of course, be seen only if the walls of the abdomen are very much relaxed. The patients themselves feel this peristaltic unrest that is caused by increased contractions of the hypertrophied muscles of the stomach. Spasmodic movements of this kind may occasionally occur, even though there be no obstruction in the pyloric region; this is particularly the case in hyperacidity and hypersecretion, and in both these conditions the patients frequently complain of these sensations. In rare instances the pylorus may become spasmodically closed, and the patients feel a spasmodic contraction of the stomach that relaxes after a time.

The appetite varies in different cases: in mild degrees it may be normal; in more advanced degrees the patients feel satiated even after eating a very small amount. The primary disease will, of course, exercise a marked influence on the appetite; in cases of ectasy, for instance, that are caused by carcinomatous stenosis of the pylorus, the appetite is usually very much reduced. Here there is a distaste for food even before ectasy itself is well developed. On the other hand, we frequently see cases of continuous secretion of gastric juice where the appetite is good or even increased, whether the ectasy is advanced or slight.

The thirst is a more important symptom than the appetite. In all cases of advanced ectasy the patients complain of increased thirst; the higher the degree of ectasy, the greater in general the thirst. This need not surprise us, for we know that the stomach cannot absorb water and that the propulsion of fluid into the intestine is greatly impeded whenever the stomach is much dilated. In all these cases we usually see a decreased diuresis, with dryness of the skin and of all the tissues. There are certain variations in this respect in the different forms of ectasy; in ectasy, for instance, that is due to a carcinoma of the pylorus, there is usually less thirst than in those cases that are complicated with hypersecretion; in general, however, we may say that an increase of thirst is an important symptom of advanced insufficiency and ectasy, and that the increase in the thirst parallels the development of the ectasy. The tongue offers no characteristic features; what symptoms are observed about the tongue are due to the primary disease. In insufficiency of the stomach the tongue may be either reddened or very much coated; it may be either moist or dry. There seems to be a tendency to dryness in cases of advanced ectasy.

Vomiting is a very important symptom of this condition; it is seen chiefly in advanced cases of ectasy that are due to stenosis of the pylorus. Vomiting itself can hardly be considered a characteristic sign, but the type of vomiting, the time at which it occurs, and the constitution of the vomit are all important. In mild degrees of motor insufficiency and in atonic insufficiency there is either no vomiting or there are very slight transitory attacks. In the different stages of ectasy, however, particularly in those cases that are due to stenosis of the pylorus and in which the stomach is constantly filled with food, there is much vomiting. Vomiting in these cases is characterized by the following peculiarities: Large masses of food are vomited at one time; the attack usually occurs very late after the meal, and occasionally particles of food that were eaten the day before or even several days before are found in the vomit. As soon as vomiting occurs the patients usually feel much relieved; after a short time, however, there is a new accumulation of food in the stomach and a new attack of vomiting. As a rule, the attacks recur at irregular intervals. Many patients produce it artificially because it relieves the feeling of pressure and tension in the stomach.

Vomiting is frequently preceded by cramp-like sensations, by a feeling of unrest in the stomach; that is usually due to efforts on the part of the stomach to get rid of its contents. I have already mentioned that this symptom is observed chiefly in stenosis of the pylorus.

Vomiting is occasionally preceded by violent pain, particularly in those cases of ectasy that are accompanied by hyperacidity; in other cases again vomiting occurs suddenly and unexpectedly, and it looks as if the stomach were simply running over.

The vomit, as a rule, tastes sour or bitter. The general appearance of the vomit will depend on the character of the secretory perversion existing. In carcinoma of the pylorus that leads to stenosis large quantities of coarse food-remnants, particularly meat-fibers or pieces of bread, are found. If there is hypersecretion, the vomit usually separates into three layers and develops a considerable quantity of gas.

Blood is quite frequently found in the aspirated stomach-contents and in the vomit in cases of dilatation of the stomach; the admixture of blood is seen chiefly in carcinoma and ulcer.

The nervous system may also be involved in advanced cases of motor insufficiency and ectasy, immaterial what the origin of these perversions. We need not be surprised to find the nervous system involved in functional disorders that are so severe that they damage the general nutrition of the patient.

Patients of this kind frequently complain of stupor, headache, vertigo, a feeling of deafness, and various paresthesias of the limbs.

These symptoms may be due to a variety of causes: they may either be due to withdrawal of water from the tissues—this would be analogous to the origin of the nervous symptoms we frequently see in violent attacks of vomiting—or they may be the result of an autointoxication from the absorption of abnormal metabolic products that are formed by the putrefaction of stagnating stomach-contents. Other authors are inclined to regard the nervous symptoms merely as signs of general inanition. All these explanations are merely hypothetic and it is hardly worth while to discuss them at length.

Another symptom that is frequently seen is so-called gastric vertigo. Patients in advanced stages of ectasy frequently complain of vertigo, stupor, and headache. Many authors assume that these symptoms are the direct consequence of the disease of the stomach, and interpret them on the basis of an autointoxication following the retention of decomposed ingesta. It is an open question whether or not this explanation is the correct one. Similar symptoms are seen in various diseases of other organs that lead to loss of strength, so that we are justified in inquiring whether these symptoms are peculiar to certain diseases of the stomach or whether they are merely the expression of a general disturbance of nutrition. This so-called gastric vertigo, at all events, possesses no distinctly characterized clinical peculiarities.

A peculiarly interesting symptom is tetany. Although motor insufficiency and ectasy are very frequently seen, tetany is rarely observed; it is most commonly seen in those forms of ectasy that are combined with hypersecretion, less frequently in the other forms of ectasy, as in carcinoma and in cicatricial stenosis following ulceration.

It is impossible to say whether all the cases that have been published so far were really cases of tetany, as the description furnished by the different authors is not conclusive. In the majority of instances, however, we are undoubtedly dealing with genuine tetany, for all the important and typical symptoms were present. Among these may be mentioned Trousseau's phenomenon, the facial phenomenon, the increase of mechanical irritability, and the quantitative increase in the electric irritability of the nerves.

Tetany is one of the most serious complications of ectasy. Of 27 cases that I find reported in the literature, 16 terminated fatally. Within the last three years I have observed 3 cases of tetany in dilatation of the stomach, and all 3 ended fatally. It is undecided how the attacks originate; Kussmaul, who was the first to call attention to this complication of dilatation of the stomach, seeks to explain tetany from the loss of water in the tissues; in other words, from abnormal dryness of the tissues. He believes that the attacks of tetany are analogous to the convulsive attacks seen in cholera Asiatica and cholera nostras. Other authors believe that tetany is a reflex process, and the majority of authors claim that it is an autointoxication from the intestinal tract. In one of my cases the attacks of tetany always occurred a short time after the introduction of the sound and after a small portion of the stomach-contents had been aspirated.

[The nature of gastric tetany cannot be said to be positively determined, although the weight of evidence is now in favor of autointoxication as the etiologic factor. The dehydration theory of Kussmaul has recent support from Fleiner,[1] but is generally regarded as untenable. The reflex theory advocated by Germain Sée is still mentioned, and while both of these conditions may contribute to an attack, most clinicians turn toward the intoxication theory as the only one that can reasonably explain the affection.

In 1891, Ferranini, quoted by Morgan,[2] reports studies on the brain of a fatal case of gastric tetany in which the degenerative alteration of the cell protoplasm apparently showed the results of toxic influences. Similar changes have been found by several other observers. The fact that the seizures most often appear soon after the use of the stomach-tube, by means of which abrasion of the gastric mucosa might have been made and thus the absorption of toxic substances from the gastric contents favored, may be interpreted as further evidence of the toxic theory.

Halliburton and McKendrick[3] have reported animal experiments in which they injected the fluid of the gastric contents into the circulation, but their results, like those of their predecessors, were rather unsatisfactory. The case from which the gastric contents was secured made a recovery after surgical intervention, a method of treatment which was first recommended by Mayo Robson,[1] who has shown that drainage of the stomach by gastro-enterostomy early in the history of gastric tetany is likely to be followed by satisfactory results. There are cases, however, in which death takes place during the first seizure, as in the case of Soltau Fenwick.[2] In this instance death occurred after sixteen hours of tetany, and shows that in some cases the attack is so serious that surgical measures would also seem to be inadvisable.—Ed.]

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[1] *Phila. Med. Jour.*. June 22, 1902, p. 1202.

[2] *Ibid.*, May 18, 1901.

[3] *Brit. Med. Jour.*, June 29, 1901.

Course of Motor Insufficiency and Ectasy.—Motor insufficiency, atony, and ectasy of the stomach, as a rule, develop slowly. In the beginning there is usually only a slight degree of motor insufficiency; provided the primary cause continues to act, this insufficiency gradually increases in severity until it finally reaches advanced degrees and leads to permanent dilatation of the stomach. In this way the pronounced picture of ectasy is created. There are a few exceptional cases on record in which the latter condition developed acutely, and while there are only very few cases of this kind reported in the literature, I believe that these forms are not so rare as might ordinarily be supposed.

The following writers have published reports on acute forms of ectasy. The first one to publish a case was Hilton-Fagge,[3] who, in 1873, reported 2 cases of acute dilatation of the stomach; in both of his cases the exact cause of the disease remained obscure. Oser,[4] in his paper, claimed to have repeatedly seen the development of positive signs of atony in cases of acute catarrh that had always enjoyed perfect digestion. Kundrat[5] reports a number of cases of acute dilatation in children. Of late years a number of cases of this character have been published—for instance, by Kelynack,[6] Schulz,[7] Boas,[8] and Albu.[9]

Both the cases of Kelynack and Schulz were reduced, cachectic individuals; the dilatation of the stomach occurred suddenly, without any demonstrable cause. The development of the lesion was exceedingly rapid, and led to the death of the patients in a short time. Boas reports a very characteristic example of acute atonic ectasy. His patient was a high-school pupil of twenty years; after a dietary indiscretion (eating large quantities of fat goose) he became very ill; there were loss of appetite and belching, but no nausea or vomiting. Diarrhea occurred on the third day, but lasted only for three days. The patient was put on a soup-diet, but nevertheless continued to vomit, the amount vomited growing more copious from day to day. Boas did not see the case until four weeks after the beginning of the disease. On examination, the stomach was found to be very much dilated, and a large quantity of stomach-contents was aspirated containing an abundant quantity of free hydrochloric acid and a large amount of sulphureted hydrogen. A course of regular lavage was instituted, and the vomiting soon stopped and the appetite improved. The patient was carefully nourished for four weeks longer, but even at the end of this time there was always some residue in the stomach at a time when the organ should have been empty.

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[1] *London Lancet*, November 26, 1898.

[2] Young, *Brit. Med. Jour.*, March 8, 1902.

[3] *Guy's Hosp. Rep.*, 1873, vol. xviii.

[4] "Magenerweiterung," in *Eulenburg's Real-Encyklopädie.*

[5] Gerhardt's *Handbuch d. Kinderkrankheiten*, vol. iv.

[6] *Medical Chronicle*, 1892.

[7] *Jahrbücher d. Hamburg. Staats-Krankenanstalten*, 1890; Leipsic, 1892, p. 145.

[8] *Deutsch. med. Wochenschr.*, 1894, No. 8.

[9] *Ibid.*, 1896, No. 7.

This case is a typical example of acute and obstinate dilatation of the stomach following an indiscretion in diet. A. Fränkel[1] reported 2 interesting cases soon after Boas published the one described above. One of his patients was a little girl of six years who was in the habit of eating a great deal. After eating a large quantity of green-peas the child suffered from indigestion that soon developed into acute dyspepsia; there were considerable nausea and vomiting that lasted for four days. Three days after the dyspeptic disturbances began considerable distention and dilatation of the stomach could be demonstrated. On the evening of the third day Fränkel found the child in a state of collapse; he learned that during the whole afternoon the patient had attempted to vomit, but without result. The girl complained of severe thirst. On examination, it was found that a loud stomach sound could be heard four fingers below the umbilicus, and that still further down there was a fluctuating swelling that corresponded to the fundus of the stomach filled with fluid.

The stomach was pumped out, and a whole liter of a brownish fluid was aspirated that contained a large number of the green peas that had been eaten four days before and had not been digested at all. Lavage was again performed the next day, and more peas were brought up; the condition of the child gradually improved. Six weeks later, however, a relapse occurred; the child recovered from this and remained well.

The second case of acute dilatation that Fränkel reports ended fatally. This patient was a servant girl of twenty-seven years who had formerly been very anemic and had been suffering from symptoms of ulcer for several years. In 1888 and 1891 there had been an attack of hematemesis. The patient came to Fränkel with symptoms of gastric hemorrhage; at the same time she was passing an abundant quantity of black blood in the stools. The diet was carefully regulated, and the condition of the patient improved very much in the first five days. Soon, however, attacks of violent vomiting occurred, and the patient died very shortly in collapse.

An autopsy was performed, and it was found that the stomach occupied a vertical position in the abdomen, the pylorus was pulled downward and drawn out, together with the pyloric antrum, so that it formed a tubular piece of intestine that was about 15 cm. long. The portion of the stomach nearest to the antrum was three fingers above the symphysis; the rest of the organ, particularly the fundus, was placed vertically. The pylorus, therefore, had been completely separated from the stomach by the horizontal portion of the duodenum. There was no trace of ulcer; all that could be found were small capillary erosions.

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[1] See *Sitzungsberichte d. Verein. f. innere Med.*, January 15, 1894; *Deutsch. med. Wochenschr.*, 1894, p. 155.

This case is interesting, not only on account of the peculiar origin of the ectasy, but also on account of the profuse gastric hemorrhages. The latter had led the physicians who had charge of the case to diagnose ulcer, whereas the autopsy showed that there were only slight capillary erosions. Cases of this kind in which there are profuse gastric hemorrhages during life, but in which the autopsy findings are negative, or at best reveal slight capillary erosions, have been frequently found, particularly in anemic subjects.

Albu's case is interesting chiefly on account of its peculiar etiology. His patient was a man of twenty-six years who entered the hospital with scarlet fever. During the period of desquamation violent attacks of vomiting occurred, the stomach became acutely dilated, the patient fell into collapse, and finally died. On autopsy, the stomach was found greatly dilated and there was parenchymatous gastritis. During life no peristaltic movements of the stomach had been observed, and just before death vomiting had also ceased, apparently because the paralysis of the gastric musculature had become complete.

The cases described above show that acute dilatation of the stomach may even terminate fatally. Another suggestive feature is the absence of vomiting in several of these cases with advanced ectasy. This can be explained only from weakness of the stomach resulting from excessive distention of its walls.

[Since the interesting case of acute dilatation of the stomach described by Pepper and Stengel[1] a number of additional cases have been reported. Julius Friedenwald[2] believes that some cases take their origin in gross indiscretion in diet. Another cause of the disease is found in a sudden paralysis of the muscles of the stomach, the result of poisoning of the nerve-centers.

I saw a case of extreme acute dilatation accompanying a fatal acute miliary tuberculosis occurring in a child eleven years old, the illness continuing but eleven days. Much interest has arisen in view of the comparative frequency with which this condition appears as a complication after surgical operations.—Ed.]

We have already mentioned that motor insufficiency and ectasy of the stomach usually develop more slowly and run a more chronic course. The development of these cases varies according to the exact type of the disease. It will be different in simple atony and in atonic ectasy than in secondary ectasy that follows stenosis of the pylorus or of neighboring parts. In general it may be said that the latter form of ectasy shows a greater tendency to progress, and, as a rule, develops into more advanced forms.

The nature of the primary disease, of course, largely determines the course of the ectasy; in carcinoma of the pylorus, for instance, the peptic powers of the stomach are disturbed so that decomposition and fermentation of the stomach-contents occur; this, combined with stagnation of the ingesta, has a tendency to increase the motor insufficiency. In cases where the secretion of gastric juice is not disturbed, the conditions for a restitution to normal are more favorable. In all cases of gastric insufficiency and ectasy, however, the character of the diet and the amount of fluid taken will exercise an important influence on the further development of ectasy. If appropriate treatment is begun early enough, insufficiency and ectasy may be kept within moderate limits for a long time, provided, of course, the primary cause of the disease is not a malignant stenosis.

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[1] *Amer. Jour. Med. Sci.*, January, 1897. [2] *Amer. Medicine*, August 10, 1901.

In primary atony the conditions for relief and cure are more favorable. Here, too, the primary cause of the disease is, of course, of paramount importance. If the stomach has been overloaded with undigestible food only once, or if the other possible causes only acted a single time, atony is, as a rule, curable, provided appropriate treatment is instituted at once. If energetic measures are not employed at once, atony may persist for a long time and even lead to permanent ectasy. In many cases of chronic ectasy a history of a sudden onset of the trouble will be elicited; generally the first symptoms are followed by remissions and exacerbations. As long as the symptoms are not severe, the patients do not consult a physician, and attempt to treat themselves by restricting their diet; or, again, they consult a physician who does nothing for them beyond prescribing a stomachic or some other remedy, simply because he takes the disease for a simple gastric catarrh. The further development of the trouble and its obstinate character will soon show, however, that the condition is more serious than is at first suspected. If cases of this kind were treated by thorough lavage and exclusive feeding by the rectum for a time, we would undoubtedly succeed more frequently than we do in preventing the development of these acute forms into chronic ones.

If the damage is inflicted for a longer period of time; if the patients habitually overeat or eat too fast—the development of these cases is different. As soon as atony has once developed, the stomach need be only slightly overtaxed in order to cause the development of atony into ectasy. If patients of this kind indulge in excesses or continue to overload their stomach with food and drink, very acute and very severe symptoms may develop; the stomach may become greatly distended and overfilled when it is atonic, and Kussmaul has shown that under these circumstances the heavy pyloric part may drop downward in the abdomen, draw the duodenum after it, and compress that part of the intestine where it is attached to the spinal column in such a manner that its lumen is converted into a narrow slit or the whole intestine is occluded. If this happens, very violent dyspeptic symptoms may naturally develop.

If the abuses I have mentioned above are continued, atony gradually increases and the stomach becomes more and more distended and ectatic.

Slight degrees of atony and even atonic ectasy frequently remain unrecognized chiefly because the symptoms produced by these lesions are not characteristic. The patients complain of thirst, a feeling of fulness and discomfort after eating; occasionally there may be an attack of vomiting. In addition a variety of nervous and neurasthenic symptoms sometimes develop.

Cases of this kind are frequently taken for neurasthenia; in other instances the disease is diagnosed as "chronic catarrh of the stomach."

The subsequent development of these forms will depend greatly on the treatment that is instituted; even cases of ectasy that are due to stenosis may be greatly improved by proper treatment. Unfortunately, it is impossible to remove the cause, and consequently all that we can do is to limit the further development of insufficiency and ectasy.

In simple atony and atonic ectasy the conditions are more favorable for a rational therapeusis. Here a well-selected diet combined with certain mechanical methods frequently yields very good results if the disease is not of too long standing; in some cases a cure may even be brought about. The quantity of urine excreted is a valuable index of improvement. We have already mentioned that in cases of advanced ectasy diuresis is, as a rule, reduced; if the excretion of urine, therefore, rises, this may generally be considered a sign of improvement of the motor powers of the stomach.

Cases of this kind frequently suffer from relapses; in general, however, the patients are responsible for this themselves, for as soon as they feel comfortable they violate the rules laid down by the physician. The best results are consequently seen in clinics and sanatoria; in private practice a rational cure can hardly be carried out, as the obstacles encountered are almost insurmountable.

From all that has been said we learn that the course of the disease may vary greatly. Mild forms of insufficiency may recover completely if the primary cause is removable. Stenosis can be ameliorated for a time, but cannot be permanently improved unless a cure can be brought about by surgical measures.

Diagnosis.—Motor insufficiency can usually be diagnosed without difficulty. It is impossible to make a diagnosis from the general disease-picture and the subjective symptoms; the only way to recognize the disease is to examine the motor powers of the stomach directly by the methods of examination we have described above.

Even the mild degrees of this condition should be recognized at an early stage if we made it a rule to examine not only the quantity of hydrochloric acid, but also the amount of residue and other factors that we have outlined above; this should be done in every case in which the stomach-contents is removed for diagnostic purposes.

I cannot emphasize sufficiently the fact that the examination of the stomach-contents possesses a much greater diagnostic value than most clinicians seem to realize. We attempt a great deal more by this examination than to settle the hydrochloric acid question. As a matter of fact, the diagnostic examination of the stomach-contents is intended to give us a complete picture of the state of digestion in the stomach. Even the most accurate quantitative determination of the hydrochloric acid is not sufficient, any more than a very careful quantitative determination of the albumin is sufficient to enable us to diagnose a disease of the kidney unless we also determined the quantity of urine, examined the formed elements of the sediment, etc. Whenever we examine stomach-contents, the quantity, the constitution, and the character of the residue, the development of gas, and many other factors must be determined. The amount of food residue that is obtained indicates the degree of motor power of the stomach; if this point is considered, even the mild forms of motor insufficiency will be recognized. In severe cases in which a large amount of residue is still present in the stomach seven hours after a test-meal the organ should be washed out in the morning in order to determine the exact degree of motor insufficiency in the manner described in preceding paragraphs.

It is an easy matter to decide what the degree of motor insufficiency is. The longer the ingesta remain in the stomach, the greater the motor insufficiency. It is essential to determine the degree of insufficiency in every case in which the existence of this perversion can be recognized. The most reliable way to do this is to examine the stomach-contents at different times; also to investigate whether succussion-sounds can be elicited at a time when they should not be heard. In the more severe degrees of motor insufficiency the stomach will not be found empty in the morning. The quantity of urine is also a valuable index; the more severe the insufficiency, the smaller, as a rule, the quantity of urine excreted in twenty-four hours. Such severe degrees of motor insufficiency, as a rule, lead to permanent dilatation, and enlargement of the organ, to ectasy.

If the examination of the stomach and its contents is carefully performed, this disease cannot be confounded with any other form of stomach trouble.

It is almost impossible to confound megalogastria with motor insufficiency; in the former disease we are dealing with an abnormally enlarged stomach, with motor powers that are sufficient. Megalogastria cannot be taken for ectasy, because in the latter condition the stomach is not only enlarged, but there are also symptoms of insufficiency. Here, too, the differential diagnosis must be made from the length of time that the ingesta are retained in the stomach. Megalogastria, moreover, rarely causes any distress to the patient.

Some authors attempt to make a differential diagnosis between nervous dyspepsia and chronic gastritis and the condition we are discussing. I need only recall the fact that motor insufficiency is not a disease *sui generis*, but merely a perversion of function. The only way in which to determine the existence of such a perversion is to employ all the methods described above. Motor insufficiency may exist alone, but it may also be combined with gastritis or with nervous dyspepsia. These two diseases can, therefore, be confounded only with motor insufficiency if the subjective symptoms of the patient alone are considered.

Whereas the differential diagnosis between gastrectasy and megalogastria is easy, a distinction is not so simple between megalogastria and gastroptosis or vertical dislocation of the stomach. If the lower boundary of the stomach is situated lower down than normal, two things may be possible: there may either be gastrectasy, or the whole stomach may be lower down in the abdomen than normal. We can diagnose the former state if the upper boundaries of the stomach are in their normal position and if the food remains in the stomach for an abnormally long time. We can diagnose simple gastroptosis if the upper boundary is lower down than normal and symptoms of insufficiency are absent. The only way in which we can make a differential diagnosis is to fill the stomach with water or with air and to determine its exact boundaries. Water may be introduced into the stomach through the sound, and the boundaries of the organ determined by percussion; the size of the stomach, and particularly the position of the lesser curvature, can be determined with greater accuracy by inflating the stomach with air or by developing carbonic-acid gas in the stomach.

If gastroptosis and ectasy occur together, the matter is more complicated. This combination is quite frequently seen, for, on the one hand, gastroptosis favors the development of ectasy, and on the other, ectasy occasionally leads to a dislocation of the whole stomach downward. In practice the matter is not so difficult as it appears. Mild degrees of enlargement of the stomach are clinically without significance. The main question to be decided is always whether or not there are symptoms of insufficiency; this question can easily be answered by examining the stomach-contents. On the other hand, it is an easy matter to diagnose ectasy when it is combined with gastroptosis, for, on the one hand, we can inflate the stomach; on the other, we will find that the organ is always full and is abnormally large.

As soon as the existence of ectasy and of dislocation or malposition of the stomach has been determined, it remains to decide what relation exists between these two conditions; that is, whether the enlargement of the stomach secondarily caused a sinking of the organ, or whether old adhesions cause bending, distortion, or contraction of the stomach, and in this way led to stenosis and secondary ectasy. Here, too, the history and the development of the case, the exact determination of the boundaries of the stomach, the position of the pylorus and the lesser curvature when the stomach is empty and full, when it is inflated and when it is flaccid, will decide this question. In many cases it is difficult to perform all these tests. If, for instance, the subject is very obese, so that the abdominal walls are thick, or if the muscles of the abdomen are very tense, it may frequently be very difficult to determine the boundaries of the stomach.

This leads to another question, which must be answered in every case of ectasy, namely, whether or not the gastric insufficiency is due to primary weakness of the muscularis—that is, whether it is so-called atonic ectasy, or whether it is due to some mechanical obstruction of the pylorus or of neighboring parts. It is of great practical importance to decide this question, for the treatment of ectasy must always be directed against the primary cause of the disease. In cases of simple atony treatment should be instituted that will raise the tone of the muscle-walls; in cases of stenosis the obstacle must be removed.

It is impossible to differentiate these two forms from any one symptom that they present; in order to decide which form is present, all the factors concerned in the case, the history of the disease, its course, and all the objective findings must be considered. In many cases it is possible to render a decision at once; for instance, if the typical syndrome of cancer of the pylorus is presented—namely, tumor, ectasy, lack of free hydrochloric acid, the presence of lactic acid, and similar symptoms. Such a typical and characteristic symptom-complex will, of course, be seen only in advanced stages of the disease, and the earlier stages of carcinoma of the pylorus are very difficult to diagnose.

We may say that in general advanced degrees of dilatation of the stomach indicate a mechanical obstruction rather than atonic ectasy; there are, however, a number of exceptions to this rule. I have repeatedly seen cases of atonic ectasy in which the stomach occupied the whole abdominal cavity. Wherever a history of ulcer can be elicited we will be more apt to diagnose a stenosis following ulcerative cicatrization than primary motor insufficiency. In continuous secretion of gastric juice it is frequently difficult to render a decision, for ectasy combined with continuous secretion of gastric juice may either be atonic or may be due to cicatricial or spastic contraction of the pylorus. The only way in which to determine the true condition of affairs is carefully to analyze all the symptoms of the case. If gastric hemorrhages are known to have occurred, we may assume that ulcer existed at some time and led to cicatricial stenosis.

I have found that peristalsis may frequently be a valuable criterion in the diagnosis of these conditions. If the peristalsis of the stomach is very active, some mechanical obstruction to the exit of the ingesta may be suspected. In order to discover peristaltic movements of the stomach the abdomen must be carefully inspected; and if peristaltic movements are absent, this does not necessarily indicate that a stenosis does not exist. In late stages of advanced dilatation the musculature of the stomach, even though it be hypertrophied, may gradually weaken, so that all peristaltic movements cease. Wherever we find very active peristalsis, we know that there is increased muscular activity; and we may know that the case is not one of primary muscular insufficiency. The most pronounced peristaltic movements are seen in cases of cicatricial stenosis of the pylorus combined with hyperchlorhydria.

A certain amount of information can also be gained from the rapidity with which water runs into and out of the stomach during lavage; the better the tone and the greater the contractile powers of the gastric musculature, the greater the pressure within the stomach. When the pressure within the stomach is increased, water will be propelled from the organ with greater rapidity and flow into the organ less rapidly than if the pressure within the stomach is normal; if the stomach is flaccid, the opposite is seen—that is, water runs in very rapidly and runs out slowly. In the latter instance a peculiar phenomenon may be observed when the water runs into the stomach; within the funnel that is attached to the stomach-tube the water may be seen to form little vortices that aspirate air into the stomach; as soon as the funnel is lowered so that the water runs into the stomach more slowly, these little whirlpools disappear. This peculiar phenomenon indicates flaccidity of the stomach-walls, a condition that is seen only in the atonic form of ectasy and never in that form that is due to mechanical obstruction; in the latter instance it may, of course, occur if the musculature of the stomach undergoes secondary degeneration.

[Palpation often affords important aid in diagnosis in case of pyloric obstruction. The pyloric end of the stomach is frequently found below the arch of the ribs, and when this is the case, any thickening of the pylorus may be felt by careful palpation. When the abdominal wall is thoroughly relaxed, an induration or even a spastic state of the pylorus may be discovered by this method.—Ed.]

Permanent regurgitation of bile into the stomach is also important from a diagnostic point of view. This accident occurs only in stenoses of the duodenum that are situated below the papilla, and never in stenosis of the pylorus. In rare cases of atonic ectasy some bile may occasionally be found in the stomach for a short time.

[Occasionally when the pylorus is thickened, and when, as a result, the tissues are rigid and inelastic, there may follow an insufficiency of the pylorus even though a considerable atresia of the pylorus exists at the same time. Under these circumstances a retrograde flow of bile into the stomach may take place.—Ed.]

The results seen from methodic treatment of these cases may also be utilized in making a differential diagnosis between the two forms. If ectasy and motor insufficiency improve rapidly under appropriate treatment, we are probably dealing with the atonic form. I have repeatedly seen a complete cure in advanced degrees of atonic ectasy. In cases of ectasy that are due to stenosis we also occasionally see marked improvement, or, at least, an arrest of the development of the disease, but we never see a complete cure.

If the diagnosis of stenosis of the pylorus or its vicinity has been made from all these different points, the question remains to be decided, what the character of this stenosis is—that is, whether it is benign or malignant; whether it is due to adhesions or to other factors.

This question can never be decided by any one symptom: the history of the case, its development, its course, and all the phenomena that are observed must be carefully taken into consideration before a decision can be rendered. It would lead us too far were we to enter into a discussion of all these different points. I need hardly emphasize that cachexia, advanced age, glandular swelling, absence of hydrochloric acid, etc., speak for carcinoma. Even the most experienced and skilful physician will occasionally encounter cases in which it is impossible to render a positive diagnosis without keeping the patient under observation for a long time.

Prognosis.—Motor insufficiency of moderate degree, provided the primary cause is not permanent, may be cured. As a matter of fact, we see cures of cases of moderate degrees of severity more frequently than is ordinarily suspected. Many persons who seem to be born with so-called weak stomachs, and who feel perfectly comfortable for long periods of time, develop a series of gastric symptoms as soon as they commit some error in diet; the slightest indiscretion in this respect frequently leads to prolonged stagnation of ingesta with all its consequences. The more careful the selection of the diet in these cases, the better the prognosis.

We have already quoted a number of examples of acute ectasy caused by overloading of the stomach. The prognosis in these cases, as we have seen, is not always absolutely favorable. The final outcome of the disease will depend on the individual; in a perfectly healthy subject the prognosis will be better than in a person who is reduced by some serious disease. It may be said in general that in all these cases the prognosis is better the sooner energetic treatment is instituted. It appears that the laity and also a great number of physicians are prejudiced against the complete withdrawal of food for several days. The only way, however, in which a rapid cure can be brought about in severe cases of this disease is to spare the stomach absolutely for a prolonged period of time.

A general prognosis cannot be made for cases of chronic insufficiency of the stomach, neither for those forms that are combined with ectasy nor those that are not; much will depend on the degree of motor insufficiency and on the primary cause of this condition. Mild degrees of atony, provided they have not existed too long, are frequently cured; advanced cases, particularly if they are combined with pronounced ectasy, are less favorable.

The prognosis, in general, is more favorable in the atonic form of ectasy than in that form that is due to some mechanical obstruction in the pylorus or its vicinity. Even in the first form, however, very much will depend on the duration of the disease and on the presence or absence of degenerative changes in the musculature of the stomach. I have seen the most favorable results, relatively speaking, in atonic ectasies combined with hypersecretion; in a great number of cases I have succeeded in improving the motor insufficiency and the ectasy of the stomach by placing the patient on a well-selected diet and by instituting a rational therapeusis. On the other hand, I have seen a number of cases of hypersecretion in which there was only a slight degree of insufficiency in the beginning, but in which an irrational mode of life gradually produced insufficiency and ectasy of high degree.

From the prognostic point of view it is particularly important in these atonic forms to institute treatment very early—this is, before the symptoms of insufficiency become serious. In order to do this the condition should be recognized very early and treated at once. We may say, in general, that the older the ectasy or the motor insufficiency, the less favorable the prognosis in regard to the restitution of muscular tone; if the muscularis of the stomach is more or less degenerated, nothing can be expected from dietetic or mechanical measures; the only method that promises relief under these circumstances is surgical interference.

The prognosis of those cases in which ectasy and insufficiency are due to some irreparable stenosis of the pylorus or its vicinity is less favorable than that of cases of atonic ectasy; the former can be treated only surgically, and here an operation may lead to permanent improvement or even a cure. All that dietetic and other measures can accomplish is to hinder the further development of insufficiency and ectasy.

Treatment.—Certain methods and regulations apply to all cases of motor insufficiency, both the atonic and the hypertonic forms—*i. e.*, those that are due to some mechanical obstruction. There are other special methods, however, that are applicable only to certain forms of ectasy and insufficiency. All that we can do in this place is to discuss those methods that are employed in the treatment of motor insufficiency and ectasy *per se.* We cannot discuss those methods of treatment that are employed in cases of secondary insufficiency and ectasy chiefly for the primary disease, as, for instance, carcinoma of the pylorus, ulcerative cicatrices, etc. For the details of the latter methods, we refer to the sections on the different diseases.

In the section on Etiology we have already mentioned that muscular insufficiency may be congenital or may be due to an irrational mode of life and an irrational method of eating. If everybody were instructed to eat a suitable diet from childhood up, this would be the best prophylactic against muscular insufficiency.

We have also seen that exhausting diseases, infectious diseases, loss of blood, anemia, etc., predispose to atonic insufficiency. We see atony following peritonitis, rapid contraction of the intra-abdominal space, repeated pregnancies, etc. In all these cases prophylactic measures must be instituted, on the one hand, and a carefully selected, very nourishing, but not voluminous diet must be given, on the other; certain mechanical supports, like bandages and binders, etc., must be applied to compensate the loss of muscular tone.

The treatment proper should be causal in the first place. If the stomach has become atonic from rapid eating or the ingestion of a voluminous and indigestible diet, all these factors must be eliminated. In many cases all that is needed to cure the atony is to avoid these irregularities, and as long as this predisposing cause is not removed, all measures directed against the atonic condition of the stomach itself will fail. In cases where exhausting diseases have produced atony the diet should be carefully selected and local treatment be instituted; where stenoses or adhesions lead to motor insufficiency and ectasy, surgical measures should be instituted. Of this we will speak in another place.

One of the first indications of the treatment of advanced degrees of insufficiency and ectasy is to prevent the stagnation of ingesta. In atonic forms of ectasy we must endeavor, at the same time, to improve the motor powers of the stomach. In cases of ectasy due to stenosis of the pylorus there is no need of increasing the motor power, because it is not reduced, but usually increased; here we must attempt, if possible, to remove the stenosis.

These considerations teach us how important it is to determine whether we are dealing with a case of motor insufficiency that is due to stenosis, or whether we are dealing with the atonic form. In both the atonic and the hypertonic forms we must endeavor to prevent stagnation of food and overloading of the stomach. In both we must carefully select a diet that will spare the motor powers of the stomach as much as possible. In many other respects, however, the general therapeusis of these two forms differs.

The treatment of motor insufficiency may be dietetic and mechanical. Among the mechanical measures we may mention lavage of the stomach, electricity, massage, bandaging, and certain hydriatic procedures.

The first indication in the treatment of motor insufficiency and ectasy is the regulation of the diet. In regard to the quantity, the first rule should be to administer as little food as possible at each meal. Large quantities of food should never be introduced into the stomach at one sitting, because this has a tendency to overload the stomach when it is already insufficient. In general, therefore, small meals should be given at frequent intervals. In selecting the diet the degree of motor insufficiency and the character of the gastric secretion should both be carefully considered. The latter, as we have seen, may vary in the different forms of motor insufficiency. The treatment will differ, for instance, in cases of atonic insufficiency or stenosis of the pylorus in which there is normal or increased secretion of gastric juice from that used in similar conditions in which the secretion of gastric juice is decreased.

Van Swieten and other authors have recommended a so-called dry diet in advanced cases of insufficiency and ectasy, and advise that as little fluid as possible be given. Von Mering[1] has shown that in a normal stomach hardly any water is absorbed, but that, on the contrary, certain substances, like alcohol, sugar, dextrin, and peptone, when introduced into the stomach cause a secretion of water into the organ. These experiments seemed to show that the administration of a so-called dry diet was rational; and in all diseases, therefore, in which the propulsion of the food and drink into the intestine was rendered difficult, as in gastrectasy, as little fluid as possible was introduced.

Certain objections may, however, be formulated against these conclusions. The absorption of fluid in the stomach is not the only factor that has to be considered; it is just as important to know how rapidly the stomach can propel its contents onward. We know, moreover, that solid food does not leave the stomach any sooner than liquid or mushy food, and while it would be irrational to administer large quantities of fluid to a patient with a flaccid dilated stomach, the same, of course, is true of large quantities of solid food. The question will have to be answered, how rapidly do fluids, on the one hand, and solid substances, on the other, leave the stomach. A large number of experiments have been performed in this direction. Von Mering[2] and Moritz[3] have shown that water introduced into the stomach is at once propelled into the intestine in jerks. If liquid and solid substances together are introduced into the stomach, the fluid portions, particularly the water, are rapidly propelled into the intestine; later, those portions of the ingesta that are of a mushy consistency are evacuated, and finally, after a considerable time, the solid portions are softened and evacuated. It was also shown that water remained in the stomach much longer if solid food was administered at the same time than if the water was taken alone.

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[1] *Therapeut. Monatsh.*, 1893, and *Verhandl. d. XII. Cong. f. innere Med.*, Wiesbaden, 1893.

[2] *Loc. cit.*

[3] *Münch. med. Wochenschr.*, 1893, No. 38; 1894, No. 41.

All these experiments, of course, apply only to the healthy stomach; at the same time, there is no reason to doubt that the same conditions obtain in a dilated stomach. It can readily be determined clinically that water is rapidly propelled into the intestine even if there is an advanced degree of motor insufficiency. When performing lavage, it will frequently be seen that the patients have a desire to urinate soon afterward, and that the urine passed at this time is lighter than that which is passed in the intervals between lavage. This seems to demonstrate that the water remaining in the stomach is immediately propelled onward into the intestine and absorbed.

From all this we may learn that the same rule applies here as in other diseases of the stomach—namely, to administer food in a form that enables the stomach to propel it onward rapidly and easily. In this way we cause the food to remain in the stomach as short a time as possible. From what we have said it is clear that mushy and liquid food fulfils this indication better than solid food. Solid food that enters the stomach must first be crushed, softened, and liquefied before it can pass the pylorus.

I need hardly mention that large quantities of fluid should never be given at one time. The chief objection against the administration of fluids is, of course, that a stated volume of liquid food is rarely as nourishing as an equal volume of solid food. Fluids that possess no nutritive value are simply ballast in the stomach; for this reason they should not be given in cases of weak stomach or in advanced insufficiency. What liquid food is administered should be introduced into the body in some other way; we will describe presently how this can be done. We do, however, know of certain fluids that possess great nutritive value; chief among them is milk; alcohol, peptone, sugar, and concentrated salt solutions are especially bad, for they cause a secretion of water into the stomach.

From a theoretic point of view, therefore, I do not think that we are justified in condemning fluid food in insufficiency and ectasy of the stomach and in feeding our patients exclusively on a dry diet.

From a practical point of view, and to judge from my personal experience, I should say that in cases of reduced gastric secretion, whether this is due to atony or to stenosis of the pylorus, fluid and mushy food should be given. In this way as little work as possible is imposed on a stomach that is suffering from secretory and motor insufficiency. As a matter of fact, cases of this kind are capable of absorbing and getting rid of liquid and mushy food more readily than of the same amount of solid food. In cases, on the other hand, in which the secretion of gastric juice is normal or increased, we must proceed differently; if the pylorus is patent, solid food, preferably meat, may be given, as the gastric juice is so abundant that it can digest meat rapidly; if, at the same time, there is stenosis, the powers of the stomach must first be determined by preliminary feeding. If the stenosis is not too severe and the secretion of gastric juice is normal or increased, a solid meat diet may be given, but no amylacea in solid form should be administered; if the stenosis is very severe, fluid and mushy food should be administered instead. I should certainly advise examining the powers of the stomach and determining the degree of stenosis by a careful analysis of the stomach-contents before prescribing a diet in these cases.

If fluid food is to be administered, large quantities should never be given at one time. Kussmaul and Fleiner have shown that relatively large quantities of fluid may be introduced into a dilated stomach without producing stagnation if the fluid is given in small portions and in intervals of one, two, or more hours to suit the peculiarities of each case. In this way patients with flaccid atonic stomachs can frequently take large quantities of fluid: as much as two liters of milk may be given each day in this manner. This, of course, does not apply to all cases, and in each individual a preliminary experiment must be made. It is an easy matter, of course, to decide whether the fluid is retained in the stomach for a long time or not. Personally, I am convinced that large quantities of fluid are well borne if given in this way, and that this mode of administration does not exercise any unfavorable influence on the development of the ectasy.

If milk administered as above is well borne, its nutritive value may be increased by adding certain substances—as, for instance, finely divided amylacea like tapioca, rice flour, wheat flour, etc. Bouillon with egg or with a little meat solution, cocoa, etc., may all be tried in these cases. If there is so severe a degree of insufficiency and ectasy that a sufficient amount of fluid cannot be introduced by mouth, it must be introduced into the colon, otherwise the tissues will lose water. Injections of water may be given; to this may be added half a teaspoonful of salt to half a liter of water; or of a little brandy (one to three teaspoonfuls) or a little wine; enemata of meat-broth and wine (two-thirds broth and one-third wine) are also useful. Before administering the nutritive enema the bowels should be thoroughly cleansed. It is possible to introduce one or even one liter and a half of fluid nourishment in this way.

In mild forms of insufficiency the injection of fluid by rectum is unnecessary. In severe degrees of ectasy, however, it is to be recommended.

There are, finally, cases of stenosis of the pylorus that are so severe that hardly anything can pass through the pylorus into the intestine. In these instances the only way to effect an improvement or a cure is to perform an operation. In order to introduce a sufficient amount of food in these cases rectal enemata must be given for a time until operation is decided upon.

It will be seen from all that has been said that it is altogether impossible to arrange a uniform diet for all the different forms of motor insufficiency. Aside from idiosyncrasies of taste and habit, the secretory peculiarities of each case must be considered in selecting the diet. The diet should be as nourishing as possible, should occupy as small a bulk as possible, and be finely divided. If the food is given in this way, the stomach is not overtaxed. When the secretion of gastric juice is normal or increased, it is easy to meet these indications; in cases of this kind a nourishing meat diet is indicated, consisting of meat of all kinds, also poultry, fish, and eggs. Vegetables should be given in small quantities only, preferably mashed; the most suitable vegetables are spinach, carrots, cauliflower, white turnips, rice, barley, and mashed potatoes in small quantities. Strong spices should be avoided.

[It may be well to speak more definitely on the question of allowing vegetables in cases of gastrectasis, for the reason that in this country they are used in such variety and form so important a part of certain meals. In advanced cases all vegetables should be interdicted. In dilatation of less degree, the tender tips of asparagus, tender young spinach, small green peas, and young string-beans may be ventured. All these should be perfectly fresh and be finely divided in preparation. Such substances as rice, barley, and tapioca should be put through the colander and blended into a homogeneous mass or given in the form of a purée. Rice, which is commonly regarded as a most wholesome cereal, is likely to be found in the stomach many hours after it has been eaten unless it is prepared as above described. This is especially true in atonic ectasy. As a usual thing such vegetables as white turnips, cauliflower, carrots, beets, and other roots that grow in the ground become a source of embarrassment to the patient unless the motor insufficiency is very slight. Potatoes demand a law unto themselves. In some individuals well-ripened potatoes, if served immediately, boiled, baked, mashed, or in the form of a purée, are acceptable and cause no trouble. If improperly prepared, potatoes are an objectionable form of diet.—Ed.]

The best form in which to give fat is as fresh butter or sweet cream.

Alcohol, unless particularly called for, is not good in atony or ectasy. I mention the well-known investigations of von Mering, who has shown that the stomach is capable of absorbing alcohol, but that at the same time an abundant quantity of water is poured into the stomach; the larger the amount of alcohol absorbed, the greater this secretion of water. In advanced forms of ectasy such a pouring-out of water into the stomach is certainly not desirable. It appears to me, therefore, that alcohol had best be avoided in these cases. Klemperer, it is true, has shown that alcohol, like creasote, is a stimulant to the motor powers of the stomach; that its effect, at all events, is more stimulating in this respect than depressing. Whether this also applies to cases of atony and ectasy is still an open question. If alcohol seems called for, it had best be given in small doses in the form of wine; beer is to be interdicted. In very severe degrees of atony and ectasy alcohol had best be avoided, particularly in large quantities.

The same rules apply to the diet in cases of motor insufficiency combined with *reduced* secretion of gastric juice, as in cases of chronic gastritis and all other diseases of the stomach in which the secretion of gastric juice is reduced.

In cases of this character the digestible varieties of meat, particularly white meat, hashed, or fish, are good. Vegetables and carbohydrates may be used in larger quantities, but should be finely divided. Fat, particularly as fresh butter, is also permissible.

In cases that are combined with continuous secretion of gastric juice the same dietary regulations obtain that we have described in the treatment of the latter secretory perversion.

As motor insufficiency and ectasy are frequently not diseases *sui generis*, but merely perversions of function that follow in the train of a variety of primary diseases, the diet will have to be regulated according to the nature of these primary diseases; in other words, the motor insufficiency and its causes must both be considered. It is hardly necessary, therefore, to formulate any fixed dietary, particularly as I have already emphasized the leading points of view.

In discussing the form in which the food should be administered, I have already called attention to the advantages presented by a liquid diet. What has been said. applies only to fluids that have great nutritive value, as milk or cream. In general, the administration of fluids should be restricted as much as possible in order to prevent too great a distention of the stomach. Water, mineral waters, and other beverages that are taken only in order to quench the thirst should be taken in small quantities, one swallow at a time. If the skin and tissues are abnormally dry; if the patient is very much emaciated; if diuresis is slight—in other words, if the tissues are losing water—fluid should be administered in the form of enemata.

As in other serious diseases of the stomach, the attempt has been made in advanced cases of ectasy to spare the stomach altogether for a long time and to administer all food by the rectum. Rössler[1] has reported some very excellent results from this method of treatment in a variety of types of dilatation.

Personally, for some time, I have been in the habit of giving nutritive enemata in all cases of advanced ectasy. In some of my cases I limit myself to the administration of fluid by the rectum. In the majority of instances the stomach is greatly relieved if a certain proportion of the food is introduced by rectum. As a matter of fact, this method of treatment, particularly in the atonic forms of the disease, frequently leads to great improvement of ectasy and motor insufficiency. In all advanced stages I can warmly recommend the administration of a portion of the food needed by the rectum. In order to do this one or two nutritive enemata should be given every day. In very severe cases

[1] *Wien. klin. Wochenschr.*, 1893, No. 30.

the treatment may be begun by feeding the patient exclusively by the rectum for a few days; after this, increasing quantities of food may be administered by mouth.

I can corroborate Rössler's strikingly favorable results. In cases of ectasy that are not very far advanced, but in which nutrition begins to suffer, a portion of the food should also be administered by the rectum, particularly if the ectasy has advanced far enough to make the administration of large quantities of food by mouth dangerous.

In regard to the composition and the nutritive value of different enemata, I must refer to what has been said in the general part of this work. Whether or not the subcutaneous method of feeding will ever be able to replace the rectal method cannot be said to-day. Voit[1] has recommended the injection of dextrose solutions, but this author's experience and my own are too limited to allow me to formulate any definite judgment in regard to the value of this procedure.

I need hardly emphasize the fact that it is necessary to control the results of the treatment and the effect of the diet by frequently weighing the patient.

In conclusion I wish to say a few words in regard to a matter that is frequently overlooked—namely, whether or not patients with advanced insufficiency and ectasy should lie down after eating or should move about. This question is quite important.

We have seen that it makes no difference whether a healthy person moves about or sits still after eating, but in the diseases we are discussing this is different; cases of this character should lie down after eating; it is wrong for them to remain up or to walk about, because thereby the stomach is distended and pulled downward and the propulsion of the ingesta into the intestine is rendered more difficult. The best position that the patient can occupy is the dorsal position or a position on the right side. Every patient, even with slight degrees of motor insufficiency, should be instructed to lie down for one or two hours at least after a large meal.

In addition to dietetic measures, certain mechanical procedures play an important rôle in the treatment of motor insufficiency and ectasy of the stomach. Among these lavage is undoubtedly the most important one. In all advanced forms of insufficiency and ectasy, whether they are due to atony or to stenosis, lavage of the stomach is an indispensable adjuvant to treatment. The good effects of this procedure are immediate, for the patients feel very much better at once. In addition, however, lavage is directly curative when used methodically. It can bring about a marked improvement of the insufficiency and ectasy, and in some instances even lead to a complete cure of the case. This applies particularly to cases of atonic ectasy that are not very old. In cases of ectasy that are due to stenosis of the pylorus the good effects are usually only transitory.

There are, undoubtedly, mild cases of atony and motor insufficiency in which lavage can be dispensed with. In many of these cases dietetic measures and possibly other methods that we will describe are sufficient to relieve the condition of motor insufficiency even without lavage. We must not forget that there are different degrees of motor insufficiency; that the condition may be very slight; that the stomach may be but slightly distended; or, on the other hand, may be distended to a colossal degree and constantly overfilled. Methodic lavage is indicated in all those advanced cases in which remnants of food are found in the morning. No one will question the advisability of this method of treatment where particles of food from the day before are found in the stomach the next morning. It is a difficult matter to decide at what stage of atony lavage should be instituted. What we wish to accomplish by washing out the stomach is to remove all excessive strain from an organ that is insufficient—in other words, to bring about normal physiologic conditions as nearly as that is possible. Under normal circumstances the stomach propels its contents into the intestine within a certain time after the ingestion of food, and it should be found empty six to seven hours after a test-meal; besides, the stomach is an organ that requires a period of rest. In a healthy subject the stomach is always empty for a number of hours during the night, and never contains food in the morning.

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[1] *Münch. med. Wochenschr.*, 1896, No. 31.

We speak of insufficient powers, of insufficiency, so called, whenever the stomach requires more time than is normal to get rid of the food that enters it. If the ingesta remain in the stomach for an abnormal length of time, they undergo decomposition and abnormal fermentative processes are the result. I am of the opinion that lavage of the stomach should be performed more frequently than it usually is. The majority of clinicians limit this method of treatment to very severe and advanced cases of motor insufficiency. I am in the habit of performing thorough lavage of the stomach in the evening, provided I find an abundant quantity of food seven hours after a test-meal; I do this even if the stomach is empty in the morning. It seems to me that it is better to remove undigested particles of food that are found in the stomach after the normal time for their digestion has elapsed before introducing new food. In this way the stomach is thoroughly cleansed and can master the new food introduced. This is certainly better than introducing more food on top of fermenting and decomposed food-remnants. Even in those cases in which the stomach succeeds in getting rid of its contents during the night it is certainly abnormally overtaxed, and the organ must suffer from this strain and gradually grow weaker and lose tone. If the stomach is relieved of this abnormal amount of work, it will rapidly regain its tone. Very much, of course, will depend on the amount of residue; if little food is found in the evening before supper, lavage is hardly necessary, but if abundant quantities of residue are present, even though the diet has been carefully regulated and other therapeutic measures have been instituted, I always prefer to perform lavage. I do this as soon as I find from 300 to 400 c.c. of food residue in the evening before supper; no harm can certainly be done by this method, and the patient will usually be much benefited.

It is impossible to state in figures when lavage is indicated. If the physician, however, will remember that the chief indication is to restore physiologic conditions, he will rarely make a mistake.

It is true, as some authors have objected, that by performing lavage a certain amount of nutritive material is removed that possibly might have been utilized, but I argue that the retention of this undigested food in the stomach leads to a loss of tone of the organ, and consequently impedes the cure of insufficiency. Whenever I remove a certain amount of food that may still possess some little nutritive value, I immediately replace it by a new quantity of food. The latter, instead of being mixed with a decomposing mass, is introduced into the stomach after it has been cleansed; in this way the loss of nutritive material is fully replaced, the stomach is not overtaxed, and abnormal fermentation and decomposition are stopped.

In regard to the best time for performing lavage, the majority of authors seem to advise early morning washings. They do this because they wish to give the stomach plenty of time during the night to utilize all the food that has been introduced. From an economic point of view this may be correct; from a therapeutic point of view, however, I think it is a precarious matter to overtax the stomach during the night, particularly if the organ is relaxed. I am in the habit of performing lavage in the evening before supper; if I find that the stomach is not empty the next morning, I also perform lavage in the morning. Washing out the stomach in the morning is, of course, useful because it cleanses the stomach once every day; at the same time it does not hinder the stomach from being abnormally overtaxed during the night, and in this sense fails to fulfil one of the most important indications of treatment.

It is useful in performing lavage, particularly if it is desired thoroughly to cleanse the stomach, to wash out the organ once while the patient is sitting up, and again when he is lying down. It will frequently be found that the wash-water comes out perfectly clear with the patient in the erect position, but contains an abundant quantity of food-remnants as soon as the patient lies down. This is seen particularly in cases of atonic ectasy. Another good plan is to instruct the patient to move his body about, for in this way the remnants of food are more thoroughly removed from the stomach. The more thorough and careful the lavage, the better; to do this requires time and patience. The only way in which to determine how long this lavage should be continued is to study the therapeutic effects. The best index is the quantity of residue found when the stomach should normally be empty. Lavage should be performed daily, and not, as is so often done, every few days; daily lavage alone will raise the tone of the stomach and cause a reduction in the size of the stomach; in other words, these good effects will be seen only if physiologic conditions are artificially established as nearly as that is possible and not if the stomach is only pumped out from time to time when it is overfilled.

Lavage is, as a rule, performed with lukewarm water alone and is continued until the wash-water runs out clear. In many cases it is good to follow the removal of the ingesta by irrigation of the stomach with certain medicaments and antiseptic drugs. The latter aid in stopping fermentation and decomposition. Even the most thorough washing with water fails to remove all fermentative organisms; this is seen from the rapidity with which fermentation again develops soon after lavage.

The following remedies have been recommended as antifermentatives: salicylic acid, boric acid, resorcin, benzoate of sodium, creasote, saccharin, creolin, and others. The best remedy for gaseous fermentation is salicylic acid in 1 to 3 pro mille solution. Others claim good results from resorcin (2 to 3 per cent. solution) and boric acid (2 to 3 per cent.); lysol (10 to 15 drops in one liter of water) has also been recommended.

All these chemical methods of treatment and lavage are useful in any form of motor insufficiency and ectasy, provided they are somewhat advanced. Electric treatment, on the other hand, is useful only in one group of cases—namely, the atonic form. In stenosis of the pylorus, even though it be complicated with ectasy of high degree, stimulation of the gastric musculature is worse than useless, for here we can actually see from the increased peristalsis that the muscles of the stomach possess sufficient power.

The majority of clinicians prefer the faradic current, and only a few employ galvanism. There is a great diversity of opinion in regard to the relative advantages of the intraventricular and extraventricular application of the current. The intraventricular current seems to be more rational, for here electricity is applied to the diseased organ direct. In practice, however, the extraventricular method is preferable, chiefly because it is more easily carried out. According to von Ziemssen's experience, the results of the intra-abdominal application of the current are no more satisfactory than those of the percutaneous application. According to von Ziemssen, large padded electrodes of some 500 to 600 cm.[1] are employed; the larger one is placed on the anterior abdominal wall, extending from the pylorus to the fundus; the smaller one is applied from the fundus to the spinal column, corresponding to the position of the stomach. The strength of the current should be regulated in such a way that active but painless contractions of the abdominal muscles are produced. The best electrode for intraventricular application of electricity is the one constructed by Wegele. This instrument is very flexible, and has this additional advantage that the stomach can be washed out, treated with electricity, and the water that is poured in siphoned out without changing the stomach-tube.

Another mechanical method that is useful, particularly in cases of atony and atonic ectasy, or even in stenosis of the pylorus, is massage. Before this method is employed a careful diagnosis must be made and the primary cause of the ectasy determined; if there is even a remote suspicion of ulcer or of some perigastritic process, massage had better be omitted. Great development of gas in the stomach is also a contraindication for its employment. Massage is intended, on the one hand, to produce more active contractions of the muscles of the stomach-wall; on the other, to aid the propulsion of the stomach-contents into the intestine. The method of performing lavage has been exhaustively described in the general part of this work, and I refer to the rules laid down there. I have never been able to see any very marked improvement in the tone of the stomach from this method. Occasionally the impression was created that the stomach-contents was propelled into the intestine more rapidly than it would have been without massage, but I have never seen any permanent results from this method of treatment.

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[1] See remarks on this subject in the section on General Considerations.

Massage of the intestines can, with advantage, be combined with massage of the stomach, for in many cases of atony of the stomach we frequently find atony of the intestine. I need hardly mention that these manipulations should be performed only by the physician himself, and never by a lay attendant.

Hydriatic measures are applicable only to atonic ectasy. The most useful methods are external douches, particularly so-called fan douches; the Scottish douche is also useful for stimulating the muscular energy of the stomach. This douche, as we have already said in another part of this work, is applied by directing a stream of water against the stomach region and changing the temperature of the water from time to time. Other hydriatic measures, like the application of cold compresses, cold sponging, etc., may occasionally be indicated and may be of some use.

The internal stomach douche has also been recommended in atony. In order to carry out this treatment a stomach-tube is used with a number of lateral openings. Rosenheim[1] has described such an instrument, and recently Salomon[2] has constructed an improved form. Irrigation is performed when the stomach is empty, and the water should enter the stomach under small pressure. Whether or not the whole gastric mucosa is really irrigated by this method is doubtful; it must be remembered that the walls of the stomach are in contact with each other when the organ is empty. Even though the stomach-tube has many lateral openings, the whole mucosa is probably not reached. It appears to me that the ordinary stomach-tube with two lateral openings performs its purpose just as well as these so-called irrigating tubes with many holes.

The internal stomach douche should be used only in mild and moderate degrees of motor insufficiency; in advanced degrees of atony and ectasy it is hardly indicated. The addition of medicamentous substances is, as a rule, unnecessary unless it is intended to treat secretory or sensory perversions of the mucosa at the same time. If the production of hydrochloric acid is reduced, a small amount of salt may be added to the irrigating fluid—a teaspoonful to one liter of water; if, inversely, its secretion is increased, silver nitrate (1 : 1000) may be added.

Fleiner claims that another very desirable effect of the internal stomach douche is stimulation of the appetite. Fleiner recommends irrigating the stomach with certain bitter remedies after the organ has been thoroughly cleansed, and claims to have seen decided stimulation of the appetite from this procedure. He uses chiefly decoctions of hops or quassia-wood or the fluid extract of condurango.

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[1] *Therapeut. Monatsh.*, August, 1892.

[2] *Berlin. klin. Wochenschr.*, 1896, No. 31.

The following rules may be added in regard to the method of performing irrigation of the stomach. The irrigating fluid should be of a slightly lower temperature than in ordinary lavage of the stomach. In the beginning the temperature should be higher than later, and should gradually be reduced to 86° F. If drugs are added to the irrigating fluid, the stomach should be afterward washed out with lukewarm water.

The method of Einhorn,[1] of irrigating the stomach with a spray apparatus, cannot be recommended for cases of atony, because a large amount of air and water is forced into the stomach and may easily distend the relaxed and flaccid walls of the organ.

Bandages are useful, particularly in those cases of ectasy in which the abdominal walls are very much relaxed. They are particularly useful in some cases in which gastroptosis and gastrectasy are present together; at all events, it is good treatment to support the relaxed organ by a suitable bandage. In some cases, of course, the patients cannot tolerate a binder because any pressure over the stomach region causes distress.

A great variety of binders and bandages have been described for this purpose. I do not think that there is any one form of bandage that is suitable for all cases. I usually have a binder or bandage fitted for each case.

Only very few drugs are employed in the treatment of motor insufficiency and ectasy. Among those remedies that have been recommended for raising the tone and the peristaltic powers of the stomach strychnin occupies the first place; it may be given either as the extract of nux vomica, in doses of 0.03 to 0.05, or subcutaneously as a solution of nitrate of strychnin, 0.1 to 10, of which two to three divisions of the syringe are to be injected. The tincture of nux vomica is less useful.

[The tincture of nux vomica is preferred by many in this country to solutions of strychnin. It is convenient because the dose can be gradually increased without changing the prescription. It is customary to give an adult ten drops of the tincture of nux vomica before meals, and to increase the dose one drop daily until the limit of toleration is reached. By following this method it is sometimes possible to give enormous doses: for instance, 50 or even 100 drops of the official tincture without producing injurious results.—Ed.]

Creasote has also been recommended for stimulating the motor power of the stomach. Klemperer[2] recommended this drug on the basis of his experiments with oil. Other experiments with the ordinary methods in regard to its influence on the motor power of the stomach have not as yet been performed. I think that the experimental evidence adduced so far is too slight to allow us to declare creasote a valuable stomach tonic. Some clinicians claim that orexin possesses the power of stimulating the tone of the stomach. I have been unable to see any such effect from its administration.

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[1] *New Yorker med. Wochenschr.*, 1891.

[2] *Centralbl. f. innere Med.*, 1891.

A great number of other remedies may be employed in cases of motor insufficiency and ectasy and may occasionally do good, but they are not given for the insufficiency, but for other disturbances that are present together with insufficiency, chiefly certain perversions of secretion, abnormal fermentation, or other disagreeable symptoms that are caused by the primary disease.

Among such remedies I must mention alkalis, for they are particularly useful in hypersecretion, perversion that is frequently seen in combination with ectasy. Alkalis, of course, are only palliative in hypersecretion, but if given at the right time, they aid the digestion of amylacea, and in this sense indirectly relieve stagnation and the gaseous fermentation that may result from it.

Certain antifermentative remedies may also be administered for the fermentation that follows stagnation. Kuhn[1] has tested a great many antifermentative drugs in regard to their effect on gaseous fermentation, and has found that salicylic acid is one of the most useful drugs in this respect. These experiments were performed in my clinic.

Other remedies that have been recommended for reducing fermentation are creasote, resorcin, menthol, salol, carbolic acid, hydrochloric acid, salicylate of bismuth, benzonaphthol, and chinosol.

[There is danger in the use of carbolic acid as a disinfectant in gastric lavage. Some patients are very susceptible even to weak solutions, and the rapidity of the absorption when employed in this way increases the danger. There are so many other useful, and at the same time safe, antiseptics that carbolic acid is better omitted altogether.—Ed.]

Cases with advanced motor insufficiency and ectasy frequently suffer from constipation.

To relieve this condition laxatives and purgatives are frequently given. If possible, it is better to avoid these drugs, chiefly because valuable nutritive material is removed from the body, and this is certainly bad in all those cases in which the absorption of the food is reduced. Purgatives may also exercise a directly deleterious effect by favoring the development of atony.

In the majority of cases a careful regulation of the diet, methodic lavage, possibly combined with massage of the stomach and intestines, will be sufficient to regulate the bowels. The patient should be convinced that it is not necessary for all persons to have a daily passage, and that many subjects only have a passage every other day and are still perfectly healthy. If a large quantity of solid fecal matter is found in the descending colon or further up, then, of course, artificial evacuation of the contents is indicated. For this purpose water enemata, possibly with the addition of a little soap or something of that kind, may be given. If this does not suffice, oil enemata may be tried.

According to Fleiner, 400 to 500 gm. of oil at body-temperature are allowed to run into the rectum with the patient in the dorsal position and the pelvis elevated. After the infusion the patient should remain in the dorsal position for one hour, or for a quarter to half an hour if the pelvis is elevated. Fine olive- or linseed-oil should be used for the injection. If no passage occurs within three or four hours, an infusion of peppermint may be injected. Frequently 150 to 200 gm. of oil are sufficient, particularly if the abdomen is massaged at the same time, to produce an abundant passage. The action of the oil is twofold: on the one hand, it dissolves portions of the fecal matter; on the other, it stimulates peristalsis. Oil enemata may be employed daily for a long period of time; as a rule, one or two oil infusions a week are sufficient. As soon as thin stools containing bile are evacuated, the enemata should be stopped.

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[1] *Zeitschr. f. klin. Med.*, vol. xxi.

If all these methods are employed, purgatives will probably be unnecessary in the majority of cases. I consider massage of the abdomen as the most important laxative measure; but massage in order to be effective must be performed by a physician who is skilled in these manipulations. I have seen many failures, but these did not result because massage was not indicated, but because it was not properly carried out. Only in case all the measures that we have described fail, should laxatives be administered, and then only the very mild ones, as rheum, tamarind, cascara sagrada, etc.

Mineral waters should not be given in cases of motor insufficiency; in fact, they are hardly ever indicated in this condition. Sometimes the perversions of secretion that are seen together with motor insufficiency may be somewhat relieved by a course of mineral waters.

If waters are used at all, the Carlsbad waters or waters of similar composition should be given, chiefly for the hyperacidity and hypersecretion that may exist. It is always better to give the salt instead of the natural water. Large quantities of mineral water are to be condemned in atony and ectasy of the stomach. In certain watering-places the patients are instructed to move about while drinking; this is particularly bad in atony.

No medicamentous treatment of vomiting is necessary; as a rule, this symptom will disappear if the diet is carefully selected and if the stomach is methodically washed out. In obstinate cases of vomiting it may be well to put the stomach at rest altogether for a few days and to nourish the patient by rectum alone. Vomiting will persist in spite of all these manipulations only in cases of very severe stenosis, and here operative procedures should be employed as soon as possible. In other cases vomiting may persist as a result of some complication—as, for instance, abnormal irritability of the stomach, etc. If this is the case, sedatives should be administered. As a rule, the latter remedies should not be given by mouth, but either subcutaneously or in the form of suppositories. Immediate benefit is obtained from codein muriat. 0.3, atrop. sulph. 0.003, aq. dest. 10.0; half to one syringeful at a time.

As we have already mentioned, patients with advanced degrees of ectasy, as a rule, suffer from thirst. As soon as the ectasy improves and the ingesta can be propelled into the stomach more rapidly, thirst is also relieved. If necessary, the craving for fluid may be relieved by opiates—Dover's powder, etc.

We have mentioned tetany above, and have stated that it is a rare though very dangerous complication. Narcotics have been recommended for tetanic attacks; as a rule, however, each single attack lasts for so short a time that these remedies can hardly exercise their effect. The chief treatment of these cases is prophylactic. According to Kussmaul's theory, the attacks are due to a loss of water in the tissues, and the indication exists, therefore, to remedy this defect. From this point of view enemata and infusions of salt should be useful; if, on the other hand, the intoxication theory is accepted, the abundant introduction of fluid is also useful, for in this way diuresis is secondarily stimulated and the elimination of toxic substances promoted. In the cases that I have had under my personal supervision I have proceeded along these lines but have failed to see any appreciable results. My cases, it is true, were particularly severe ones.

Operative treatment of dilatation of the stomach should, of course, be considered only if all the other methods that we have described fail to give relief. Surgical measures may be advised for two purposes: first, to remove the cause of the insufficiency and ectasy—for instance, to cure the stenosis in cases of stenosis of the pylorus; second, to remove the sequelæ of insufficiency and ectasy—that is, chiefly the stagnation of stomach-contents—by facilitating the exit of stomach-contents into the intestine.

The first method is chiefly indicated in stenosis of the pylorus, whether the stenosis is due to carcinoma, is cicatricial, or is due to hypertrophy of the musculature of the pylorus, to compression or contraction from without, to adhesions, etc. The second method is indicated if the primary cause of the disease cannot be removed. A third method might be employed in some cases in which there is dilatation of the stomach—namely, the reduction of the size of the stomach by operative procedures. We will describe the different operations later on.

The first question that the clinician must answer is, When should a patient with motor insufficiency and ectasy be advised to undergo an operation. As a rule, this time can be readily determined; operative interference is indicated whenever the above-mentioned dietetic and mechanical measures fail to accomplish anything; in other words, when all efforts directed toward introducing a sufficient quantity of nutritive material fail. It is at this time that the internist should give way to the surgeon. In some cases, however, the matter may not be so simple as it appears, and it may be difficult to decide just when the operation should be performed; the best indices are the weight and the general state of nutrition of the patient. The quantity of urine is also important. If the patient does not absorb a sufficient quantity of nutritive material, even though the diet be carefully regulated, both quantitatively and qualitatively, if the ectasy and the insufficiency do not decrease, but, on the contrary, increase even though mechanical treatment be energetically instituted, and if, finally, the patient continues to lose weight, then an operation is indicated. It is best, however, not to operate too late—that is, at a time when the patient has lost a great deal of strength.

There is another class of cases in which the patient feels comparatively well as long as methodic treatment is carried out and the diet is very carefully regulated, and in which the condition of ectasy may even improve slightly, but in which we also know, from the nature of the disease, that the patient cannot be completely cured within a reasonable time; the patients, in other words, feel comparatively well only while they are undergoing treatment and while they adhere to a rigid diet; any deviation from the prescribed mode of life immediately leads to an aggravation of their condition. A great deal will also depend on the social condition of the patient. Every physician can recall cases in which the patients felt comparatively well as long as they remained in the hospital; here the ectasy seemed to improve, and if the stomach-contents was removed for diagnostic purposes it was found that the amount of permanent residue gradually decreased. As soon as the patients left the hospital, however, the improvement stopped and their condition became aggravated, chiefly because they were forced to earn their livelihood by manual labor and were forced to eat a less carefully selected and less digestible diet. These cases, as a rule, return to the hospital within a short time; usually their condition is worse than it was when they were received for the first time. It is altogether impossible for these patients, owing to their social position, to take care of themselves and to procure the necessary kind of food. If these cases are not relieved by an operation, the disease will progress and the patients will ultimately succumb to it. In cases of this character it is not the severity of the disease *per se* that calls for operative interference, but the fact that these patients are altogether unable to treat themselves by dietetic or mechanical methods in the way in which they should be treated.

If the patients happen to be of a different class, the operation would probably be postponed or possibly be omitted altogether.

We see, therefore, that it is necessary to weigh carefully all the circumstances of the case in each individual patient, and that very many factors will have to determine the exact time at which the operation should be decided upon.

Stenoses of the pylorus of all kinds most frequently call for operative treatment. We have stated above that this operation is performed in order to remove the primary cause of the ectasy and insufficiency. In cases in which the disease is not very far advanced a radical cure may frequently be brought about in this way. There are a number of operations that can be performed, the details of which cannot, of course, be discussed in this place. I will mention only Loreta's digital divulsion of the pylorus, resection of the pylorus, pyloroplasty, and the separation of adhesions.

If operations of this kind are performed at the right time, the chief result of the primary disease, namely, the stagnation of ingesta, will stop. This is the chief aim of the different operations, and may be accomplished in various ways. The character of the stenosis will frequently decide what operation shall be performed. Loreta's digital divulsion of the pylorus can, of course, be performed only in benign stenoses. In this operation the stomach is opened and the stenosed pylorus forcibly stretched by introducing one or several fingers into the lumen of the pylorus. This operation, however, has never become popular, at least not in Germany. It is not devoid of danger; it does not preclude recurrence, and can frequently not be performed even in benign stenosis. The operation of pyloroplasty that was introduced into surgical technic by Heinecke and Mikulicz is a more suitable one; it consists in splitting the stenosed pylorus longitudinally, in compressing the pylorus so that the longitudinal incision becomes a tranverse one, and then uniting the edges of the wound by sutures. The chief disadvantages of this method are that it cannot be carried out in many cases, particularly where there are solid and hard adhesions that transform the pylorus into a stiff and unbending tube. The operation *per se*, however, seems to be planned on very rational lines.

In carcinoma of the pylorus, resection of the pylorus, if it can be performed, is the most radical operation, for it not only removes the stenosis, but also the carcinoma. This operation is also useful in cases of ulcerative stenosis and stenosis from cicatricial contractions. The operation is technically very difficult, and there are, in addition, a number of cases in which it cannot be performed—if, for instance, solid adhesions have been formed with neighboring organs. Another objection to the method is the fact that in those cases where it is performed in the later stages of the disease the musculature of the stomach is so overdistended and paretic that the tone of the stomach is not improved and the stagnation of contents not prevented, even though the stenosis be cured. For all these reasons the other operation mentioned above —namely, the direct relief of stagnation—is growing more and more popular. The operation of gastro-enterostomy answers this purpose; an anastomotic connection between the stomach and the duodenum is created, and in this way food flows into the intestine through a new passage. This operation is indicated wherever the other operations that we have described cannot be performed or are not indicated. It has this advantage, besides, that it is not so radical an operation and certainly constitutes a less severe inroad than resection. The operation is useful both in malignant and benign stenoses. It can also be performed in any form of ectasy, and is not limited to those forms of ectasy that are due to stenosis. As the chief object of the operation is to relieve stasis, it is indicated even in the atonic forms of ectasy, particularly in those cases where internal treatment fails. So far the operation has been performed only a few times in the latter form of ectasy.

The third method that we employ is to reduce the size of the enlarged stomach; Bircher has devised a method for this purpose. The operation of Bircher is, of course, applicable only to cases of atonic dilatation. The stomach is sewed into folds and in this way its volume decreased. The operation will, of course, be useful only in those cases in which the stomach still retains some of its tone, and it is, of course, impossible to know in advance whether or not the stomach muscles still retain a certain tone; as a matter of fact, we hardly expect to find much muscular tonicity in those cases of atonic ectasy in which this operation will be performed; for this reason chiefly the operation has not become popular.

These are the surgical methods that may be employed in these cases. In many instances it is impossible to decide in advance which procedure is the most suitable one. In many cases the surgeon will be forced to change his original plan after the abdominal cavity has been opened. Notwithstanding great progress in the diagnosis of stomach-diseases, there are a number of cases in which an operation will be undertaken and in which it will be found afterward that the surgical procedure that was originally planned cannot be executed after all; consequently many surgeons are forced to perform gastro-enterostomy, an operation that is less severe, more easily executed, and is, in a measure, a radical operation for motor insufficiency, inasmuch as it relieves the stagnation of ingesta.

[For the relief of gastroptosis the operation of gastroplication has been occasionally practised. Besides this, Stengel and Beyea have shortened the gastrohepatic omentum by a series of ligatures. Recently the latter has advocated[1] shortening the gastrohepatic and gastrosplenic ligaments. Still others have attempted to fix the stomach in a more elevated position by means of a series of stitches attaching the lesser curvature of the stomach to the abdominal wall. Slenk[2] reports a case in which he sewed the lesser omentum to the pancreas. Last of all, Coffey[3] describes 2 cases in which he sewed the greater omentum to the anterior abdominal wall by means of a series of interrupted sutures, so placed that the omentum acted as a hammock in which the stomach was suspended. In his first case he used three sutures, in the second eight, and he now recommends a larger number, making a line of support across the omentum eight or ten inches in width. The results which have followed in the reported cases appear to be flattering, and further observations will be awaited with interest.—Ed.]

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[1] *Medical Society, State of Pennsylvania*, September 16, 18, 1902.

[2] *Centralbl. f. Chir.*, September 20, 1902. [3] *Phila. Med. Jour.*, October 11, 1902.

CHANGES IN THE POSITION AND THE FORM OF THE STOMACH: GASTROPTOSIS, ENTEROPTOSIS, HOUR-GLASS CONTRACTION OF THE STOMACH.

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Compare, besides, the well-known text-books, handbooks, and monographs on diseases of the stomach.

Introductory Remarks.—Dislocation of the abdominal organs has been known to pathologic anatomists for a long time. More than forty years ago Virchow called attention to this occurrence, and numerous investigators after him have shown that in many adults the abdominal viscera are dislocated, particularly the intestines. Clinicians, on the other hand, have not paid much attention to the changes in the shape and position of the abdominal organs, particularly of the stomach, until quite recently. A few isolated reports, it is true, may be found scattered through the literature.

Kussmaul was the first to call attention to a variety of changes in the form and position of the organ and to point out the connection existing between these abnormalities and certain clinical symptoms. This author was a pioneer in this field, as in so many other fields of medical research.

General interest in these postural anomalies was aroused by Glénard's researches on enteroptosis. Glénard was the first to call attention to the frequent occurrence of anomalies in the position of the abdominal viscera and to point out the clinical significance of this finding. He also pointed out the intimate connection that exists between enteroptosis and nervous dyspepsia. When his report appeared, the anatomic basis of a large number of nervous gastro-intestinal troubles seemed at last to have been discovered. Glénard believed that relaxation of the hepatocolic ligaments always was the starting-point of enteroptosis; this relaxation, he thought, led to ptosis and even bending of the transverse colon. Secondarily, he argued, other ligaments would become relaxed and in this way the stomach, the liver, the kidneys, etc., would become dislocated.

It may be well in this place to mention that Glénard's discoveries, however noticeable the facts described may have appeared, soon aroused a large amount of opposition. His interpretation of the genesis of enteroptosis was vigorously combated. In Germany Ewald, in particular, carefully investigated enteroptosis and the causes that led to it and placed himself strictly in opposition to Glénard's views.

It would lead us too far were we to enter into the details of all the reports that have been published on this subject. I will limit myself to mentioning some of the work of Meinert. This author, by a large number of exhaustive investigations, demonstrated that enteroptosis is by no means rare and that it occurs with particular frequency in women; he also showed that this condition is frequently met with in chlorosis. Meinert even went so far as to claim that gastroptosis was one of the most prolific causes of chlorosis; the majority of authors—and I include myself—do not agree with this view.

Nearly all modern writers seem to believe that postural anomalies of the stomach can be made responsible for many so-called nervous stomach-symptoms. We will refer to this matter later on. In this place we must first describe the different possible anomalies of position that are seen and their significance in practice. It should, above all, be remembered that the stomach alone is rarely dislocated, but that other abdominal viscera are usually displaced at the same time; the same applies to changes in form. The very nature of the diseases we are discussing includes a general involvement of the whole abdominal contents; not of the stomach alone, but also of all the organs that are in close proximity to it.

Etiology.—Even under physiologic conditions any distention and overfilling of the stomach will cause a change in its shape and in its size; consequently the stomach, when it is enlarged, must occupy more room, and at the same time will force other abdominal organs to accommodate themselves to this change in its form and position.

Analogous conditions are seen whenever the colon is either overfilled or overdistended. We also see a marked dislocation of the different abdominal viscera at the time of pregnancy and after delivery. During pregnancy not only the abdominal organs, but also the thoracic organs, become compressed to a certain extent and must change their position. This is naturally due to the fact that the enlarged uterus occupies a large portion of the intra-abdominal space that is ordinarily given to the different abdominal viscera. After delivery the opposite effect is, of course, seen.

Dislocation of abdominal organs is naturally chiefly seen in pathologic processes within the abdominal cavity; less frequently in similar processes in the thoracic cavity. A large pleuritic exudate on the left side, for instance, will push the heart to the right, and at the same time force the diaphragm downward on the left side; the natural consequence of this leads to a moderate degree of dislocation of those organs that are in immediate contact with the diaphragm, chiefly the stomach, the transverse colon, and others. The same occurs after the exudate is absorbed, particularly if the lung is unable completely to fill out the space that is now placed at its disposal. In this instance neighboring organs must be utilized to fill out this space. The affected side of the thorax will collapse, the diaphragm will move upward, and the neighboring organs in the abdomen will follow in the same direction.

Morbid processes that occur in the abdominal cavity and that lead to enlargement of different organs, so that some of the intra-abdominal space is occupied in an abnormal manner, lead to dislocations and changes in the form of other organs, particularly the stomach. Another factor, which we will refer to later on, causes dislocation of abdominal organs, namely, the irrational clothing that women adopt nowadays, and that, as we know, compresses the lower half of the thorax.

Even abnormal distention of the stomach itself, which persists for a long time, may cause a dislocation of the organ. We frequently find that the stomach in advanced stages of ectasy is dislocated downward and at the same time changed in form. The change in form varies according to the primary cause of the ectasy. In stenosis of the pylorus, for instance, the pyloric portion of the organ is usually hypertrophied and contracted, chiefly because this part of the stomach has to perform the greatest portion of compensatory muscle work. In atonic ectasy, on the other hand, these portions of the stomach are in an exactly contrary condition—namely, abnormally distended and enlarged (ballooned).

Another prolific cause of dislocation and change in the form of the organ is found in certain inflammatory processes and adhesions with neighboring organs that form as a result of these inflammations. We see such conditions, for instance, in carcinoma and after peritonitis.

We learn from all this that anomalies in the position and the shape of the stomach may originate from a great variety of causes.

The Different Anomalies in the Position and the Size of the Organ and their Symptoms.—The anomalies in the form and the size of the organ are less important from a practical point of view than the anomalies in its position.

Anomalies of form are frequently congenital, but I will not enter into a discussion of these lesions in this place. They have been called antrum cardiacum and forestomach, and both, properly speaking, are anomalies of the esophagus. Forestomach, so called, is really a dilatation of the lower end of the esophagus situated immediately above the diaphragm. Antrum cardiacum, on the other hand, is a sacculated diverticulum of that portion of the esophagus that is situated below the diaphragm. It appears that both anomalies are quite frequently encountered. In many cases they produce no symptoms; in others again coarse particles of food may become lodged in the forestomach or the antrum cardiacum and cause very serious symptoms.

Another congenital anomaly of the stomach, which has so far been observed in only a few cases, is congenital narrowing of the pylorus. As a rule, this stenosis is due to muscular hypertrophy of the circular muscularis of the pylorus. Narrowing of this character may interfere seriously with the ingestion of food or may render it altogether impossible, so that infants afflicted in this way usually succumb within a few days after birth. In other cases, again, this stenosis leads to more or less severe degrees of dilatation of the stomach. Many changes in the form of the organ result from changes in its position. We will refer to this later on, when discussing the changes in the position of the stomach.

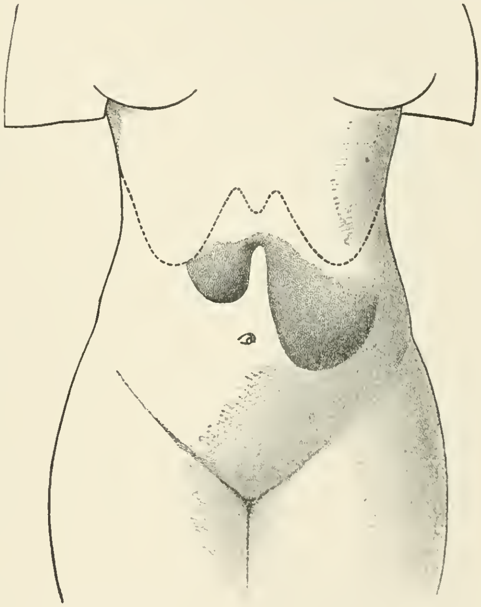

FIG. 16.—Hour-glass contraction of the stomach, according to Schmid-Monnard.

In this place we will discuss only a rare anomaly of form which is clinically of great significance, namely, so-called hour-glass contraction of the stomach. This condition may be either congenital or acquired; more frequently it is acquired. The stomach in this form assumes a peculiar sacculated outline, and is divided into two parts—a cardiac and a pyloric sac. In some instances the cardiac sac is larger; in others, the pyloric one (compare Fig. 16, which represents a case of hour-glass contraction of the stomach reported by Schmid-Monnard).

The acquired form of hour-glass contraction of the stomach is usually caused by strands of cicatricial tissue that follow an ulcer. Less frequent causes are peritonitic adhesions, corrosive gastritis, and carcinoma. I have observed a case of the latter character myself, and have described and illustrated it in the section on carcinoma. Some authors claim that this condition may originate from tight lacing.

Slight forms of this condition produce no symptoms, or at best symptoms that are not characteristic. In advanced cases the stomach may be very much dilated, and under these circumstances the division of the organ into two, usually unequal, parts, separated by a shallow or a deep division, can be recognized. It is easier to palpate the organ and to recognize the peculiar division by inflating the stomach either with carbonic acid gas or with air. Sometimes it occurs that only one-half of the stomach is distended by this method. This occurred, for instance, in a case reported by Bouveret; here the cardiac portion alone was inflated, whereas the pyloric portion was not distended; the latter was, nevertheless, found to be very much dilated, and under favorable conditions succussion sounds could be elicited over it.

The most disagreeable feature of hour-glass contraction of the stomach is the fact that the greater curvature is pulled upward so that the food, so to speak, has to climb a hill. This, of course, renders the elaboration and propulsion of the ingesta more difficult; the muscularis of the stomach is overtaxed by the excessive labor of raising the food over the inclined portion of the stomach-wall, and ultimately becomes paretic, so that those portions of the stomach that have been most seriously overtaxed become ectatic.

The size of the opening communicating between the two divisions of the stomach varies greatly. In some instances it was found to be merely a narrow canal.

The symptoms, of course, vary according to the form of the stomach, the lumen of the opening between the two divisions, the degree of ectasy, and other conditions. There are, however, a number of symptoms that are important for the diagnosis of hour-glass contraction of the stomach. It may be considered characteristic, to a certain degree, if we succeed in eliciting distinct splashing over the stomach, when the organ is apparently empty and when no stomach-contents can be procured by aspiration. This, for instance, occurs in cases in which the pyloric division of the stomach contains the material that gives the splashing, whereas the stomach-tube, of course, enters the cardiac division.

Another conspicuous symptom that some authors claim to have observed is noticed during lavage of the stomach. It may occur that nothing but clear water runs out for a time, then suddenly particles of food appear in the wash-water. I am hardly inclined, however, to attach too much importance to this latter symptom, because it may also occur in cases of advanced ectasy in which there is no hour-glass contraction of the stomach. Another peculiar symptom is occasionally observed in hour-glass contraction of the stomach, namely, that more water may run out during lavage than was allowed to flow in.

[In occasional cases the use of the stomach-tube may enable us strongly to suspect an hour-glass contraction. If, after having emptied the first portion of the stomach, the abdomen is carefully manipulated, it is sometimes possible to force backward from the second portion into the first portion of the stomach gastric juice which is different in quality from that which was previously obtained. I have sometimes succeeded, by careful manipulation, in passing the stomach-tube through the narrow opening of the constricted portion into the second part of the stomach, and obtaining therefrom gastric juice of such different character that a division of the stomach into two parts was demonstrated.—Ed.]

All these criteria, however, may fail to lead to a diagnosis, and many of these symptoms, of course, may be altogether absent.

It is quite natural that advanced degrees of this condition lead to serious disturbances of nutrition, and may finally cause the death of the patient. In a few cases a peculiar rotation of the stomach around its own axis has been observed.

The only way in which to cure this condition is by an operation. The exact method of surgical procedure will, of course, vary according to the peculiar conditions present in each individual case; in some instances resection, in others ligation, of the communicating portion and gastrorrhaphy or gastro-enterostomy are necessary. In many cases the gastro-anastomosis described by Wölfler is the most suitable method. I refer to the text-books on surgery for the details of all these questions.

There are two forms of anomalies in the size of the stomach. The organ may be either too large or too small; in the former instance we speak of megalogastria; in the latter, of microgastria. Neither condition causes any symptoms *per se*, provided the stomach is otherwise intact. In megalogastria, it is true, the stomach on inflation will be found to be large, but its motor functions will be perfectly normal; this differentiates it from an ectatic stomach, for in the latter we have permanent abnormal distention and a reduction in the motor powers of the organ.

Microgastria is occasionally seen in subjects who have been in the habit of taking very little food for a long time. Both conditions, megalogastria as well as microgastria, are, as a rule, discovered accidentally.

Anomalies in the position of the stomach are much more important and much more frequent than any of the changes we have described so far. The stomach may be dislocated in different directions—upward, laterally, or downward.

It is usually the fundus of the stomach that is dislocated upward. This accident occasionally occurs after the absorption of a pleuritic exudate of the left side, after contraction of the lung, in fact, after any process that is accompanied by a dislocation of the left half of the diaphragm upward into the thorax. It is also seen in processes that lead to great distention of the abdominal space and in this way force the diaphragm upward. Such conditions are, for instance, meteorism, pregnancy, ascites, and tumors of the abdomen. [Among the rare conditions that may lead to dislocation of the stomach upward is diaphragmatic hernia. When present, it may give rise to considerable embarrassment in diagnosis, as the colon drawn upward into the region usually occupied by the stomach may deceive the clinician unless, at the same time, he makes a very careful examination of the thorax.—Ed.]

The stomach in all these conditions may be smaller throughout or only in its pyloric portion. The fundus, at the same time, may even be dilated. According to Fleiner, it may happen that the fundus is pushed upward and to the left so forcibly that the cardiac end of the esophagus becomes bent when it passes through the diaphragm. In this way it may occur that the lumen of the cardiac end of the stomach becomes completely occluded, chiefly from below. When this occurs, the act of swallowing will, of course, not be impeded, but, on the other hand, it will be almost impossible for the patient to belch or to vomit, particularly when he is lying down (Fleiner). Lacing or wearing a belt around the waist may occasionally force the stomach upward.

Dislocation of the stomach laterally is rare. Slight degrees of lateral dislocation may be caused by large tumors of the spleen, by the left colic flexure if it is situated high up or is very much distended, or, on the other hand, by the pressure that the liver may exercise on the stomach in case it is enlarged and dislocated downward. The latter accident is, however, usually accompanied by a dislocation downward of the stomach.

The most important form of dislocation from a practical point of view, and the most frequent one, is dislocation of the stomach downward —gastroptosis. The stomach may either sink downward in its totality or be placed more vertically, or even assume a looped form. Kussmaul was the first to call attention to these different possible forms of dislocation of the stomach.

Every human being is born with a vertical stomach. During the first weeks of extra-uterine life the stomach then assumes the position that Luschka designates as normal (Meinert). This change in position is brought about by the weight of the food that is introduced into the stomach and by the action of the diaphragm. Meinert, to whom we are indebted for the most exhaustive investigations into this question, never succeeded in finding the stomach in an abnormal position in infants after they had been taking food for some little time. He expresses the belief that the stomach rarely remains in the position that would correspond to the one it occupies during fetal development. This view is in opposition to the one enunciated by Kussmaul, for the latter author explains the occasional occurrence of vertical stomach in adults on the basis of arrested development.

Meinert's investigations revealed the fact that this anomaly in the position of the stomach is more frequently found as children grow older, and is particularly frequent in girls. He examined some 50 girls of about twelve years, and found that nearly every alternate girl showed dislocation of the stomach. In the adult female patients of his gynecologic private clinic he found anomalies in the position of the stomach in more than 80 per cent., whereas he estimates that this anomaly is found in only 5 per cent. of the male population.

Gastroptosis is found in adults of both sexes, chiefly in paralytic thorax, in chicken-breast, in funnel-breast, in cases where the diaphragm or the liver is dislocated downward, and in cases where the liver is enlarged. In women another factor seems to play an important, probably the most important, rôle—namely, the clothing. Lacing and wearing skirts that compress the waist lead to compression of the lower part of the thorax, and in this way cause dislocation of the abdominal viscera, particularly dislocation downward of the stomach.

[While there can be no doubt of the importance of the wearing apparel when improperly supported as an active factor in the development of gastroptosis, there is much evidence in support of the views of Glénard that there is a constitutional defect in certain individuals as to the strength and supporting power of the mesenteric tissues. The contributions of Stiller as regards the frequency of gastroptosis when there is found present the floating tenth rib are also confirmatory of this view. Strauss[1] divides gastroptosis etiologically into two classes: The first class depends upon anomalies of body formation, and the second depends upon local mechanical causes. The majority of cases belong to the first group, and in such individuals he believes there is a characteristic abnormal formation of the thorax and pelvis; in other words, a natural predisposing condition. The fact is that in many of these cases there is a congenital delicacy or fragility of tissue, which, under strain and in the absence of support, yields, and the viscera easily become displaced.—Ed.]

Dislocation of the stomach and of other abdominal viscera is, therefore, usually acquired, and is due chiefly to the change in the normal arrangement of space in the lower half of the thorax and the upper portion of the abdominal cavity. The stomach and the other abdominal viscera must adapt themselves to these changes and to this decrease in the normal amount of space placed at their disposal, and are consequently forced to move in the direction of least resistance and to take up a position wherever they find room.

Dislocation of the stomach downward is seen in two main forms—*i. e.*, the whole organ may be dislocated downward or the stomach may occupy a vertical position. The former state is called gastroptosis proper.

Strictly speaking, we rarely see dislocation of the whole stomach downward, for the organ is fixed at its cardiac end. The fixed point is situated in the region of the twelfth thoracic vertebra, and this portion of the organ usually remains in contact with the diaphragm, even though the rest of the stomach becomes dislocated. Of course, if the stomach is heavy and dislocated downward, this portion of the organ may also be forcibly pulled from its normal position. All other portions of the stomach, however, are freely movable and can readily change their position. In this sense Meinert is correct when he denies the possibility of a dislocation of the stomach *in toto*.

The type of dislocation of the stomach will be determined by the position of the pylorus and of the lesser curvature. In some instances there will be simple sinking of the stomach; in other cases the organ will occupy a vertical position; in still others it will assume a looped shape, etc.

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[1] *Boas's Arch.*, vol. vi., No. 1.

In determining the abnormal position of the stomach clinically, the location of its lower boundary is not so important as the location of the pylorus and the lesser curvature; only where the latter portions of the stomach are dislocated downward can we speak of dislocation downward of the stomach—of gastroptosis.

The determination of the position of the lower boundary of the stomach alone is without value for the diagnosis, for the fact that this portion of the organ is lower down than normal does not prove that we are dealing with a case of gastroptosis. In any stomach that is abnormally large but still remains in its normal position, dislocation downward can be simulated.

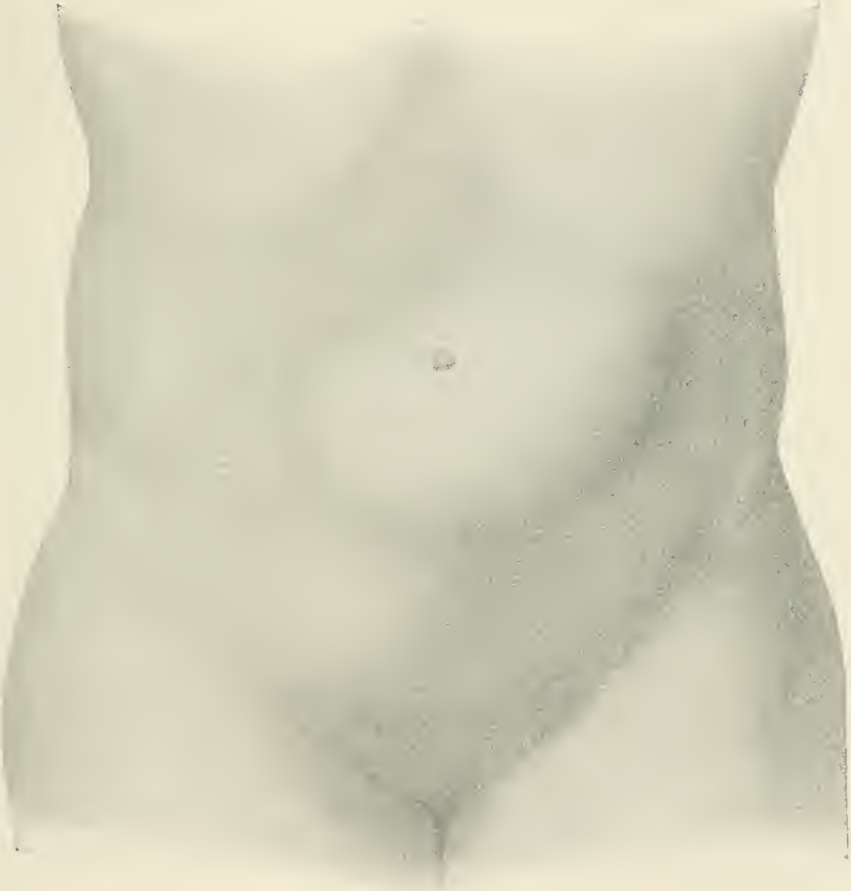
Normally, the pylorus is hidden under the right costal arch and is situated approximately on the same horizontal level as the xiphoid process. From this point the upper boundary of the stomach merges directly into the lesser curvature. For this reason the latter can never be determined when the stomach is altogether normal; for even if a normal stomach be inflated, the whole epigastric region will be uniformly distended and the area of distention will be bounded below by the greater curvature of the stomach.

In gastroptosis this is different; here the upper portion of the epigastrium is not distended, because the stomach is not there. If the organ is inflated, a depression will be seen in the epigastric region, below which there will be a bulging portion. If this protrusion is carefully examined, it will be found that its form corresponds to that of the stomach, so that the dislocation of the organ can usually be diagnosed by this simple means.

In advanced cases of gastroptosis the upper boundary of the stomach may be found in the region of the umbilicus. The lesser curvature, which usually appears as a line curved concavely upward, may either appear as a concave line running diagonally from left to right and downward, or as a vertical line, or again as a loop. The position of the greater curvature will, of course, vary according to the position of the pylorus and of the lesser curvature. Whether or not the stomach assumes a vertical position, a subvertical position, or any other position, the fact that the upper epigastric region contains no portion of the organ is considered pathognomonic for gastroptosis. The lower boundary of the organ must, of course, always be found lower than normal, even though the stomach itself be not enlarged.

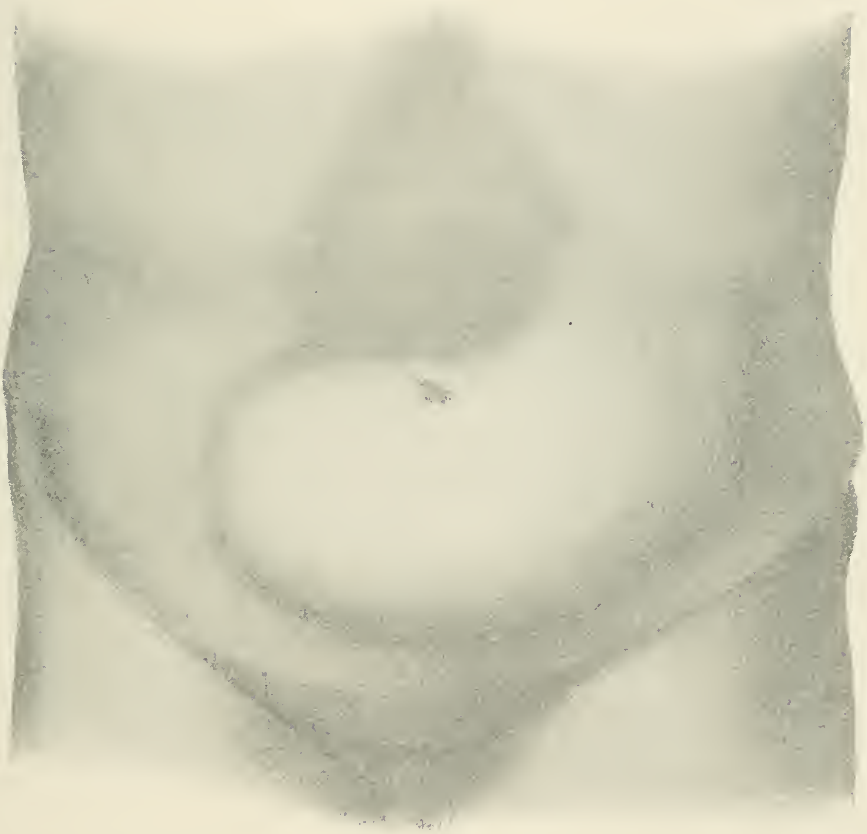
As a rule, however, gastroptosis is complicated by a more or less advanced degree of dilatation of the stomach. To judge from my own personal experience, ectasy of the stomach plays an important etiologic rôle in the causation of gastroptosis. We frequently, though not always, find ectatic stomachs dislocated downward. In certain cases, of course, in which gastroptosis and ectasy are found together, it may be a very difficult matter to decide which of the two conditions is primary and which secondary. If, on the one hand, abnormal overloading of a stomach that is ectatic may cause the organ to sink down in the abdomen, gastroptosis *per se*—and this probably occurs quite frequently

PLATE I.



Gastroptosis (Moderate Vertical Dislocation) in a Girl of Twenty-eight Years

PLATE 2.



GASTROPTOSIS WITH ECTASY FOLLOWING ULCER-SCAR CAUSING STENOSIS, IN A MAN OF FIFTY-SIX YEARS.

—may, on the other hand, render the exit of ingesta from the stomach into the intestine more difficult and thus gradually lead to a distention of the organ and to ectasy. There are many cases, however, of gastroptosis in which there is no ectasy. If the position of the stomach is carefully investigated in young persons, particularly in women, it is surprising to find gastroptosis in a great many instances where no stomach symptoms whatever were complained of, and in which a careful examination of the secretory and motor powers of the organ revealed normal conditions.

The position and the form of the stomach, when it is dislocated downward, may vary greatly in individual cases. Very much will naturally depend on the distribution of space within the abdominal cavity. The stomach will always be dislocated in the direction of least resistance. Compression of the lower half of the thoracic aperture—for instance, by lacing or wearing corsets—will naturally lead to dislocation of all the organs that are exposed to this increased pressure downward. The epigastric portion of the stomach, the pylorus, and the lesser curvature, above all, will be forced downward unless, of course, the tone of the abdominal muscles is sufficiently great to counteract the pressure from above. It appears, however, that the latter is never sufficiently powerful to do this.

Dislocation of the stomach and of other abdominal organs in different directions is seen in advanced cases of kyphosis and kyphoscoliosis. Large tumors of the liver, leukemic enlargements of the spleen, may cause lateral dislocation of the stomach and at the same time dislocation of the organ downward. The position of the stomach will vary according to the direction of the greatest pressure.

The most important cause, however, of dislocation downward is, as we have said, lacing, and, in general, the present customary dress of women. Wearing skirts that are attached around the waist is almost as bad as wearing corsets. Great relaxation of the abdominal walls; a sudden decrease in the contents of the abdominal cavity—may all lead to gastroptosis. Relaxation of the abdominal muscles, it is true, may, to a certain extent, be compensated by the greater distention of the intestines that usually follows; complete compensation is, however, rarely seen. For this reason, for instance, we frequently see gastroptosis develop in women who cannot take sufficient care of themselves during the puerperium; or if gastroptosis was present in women of this kind, we frequently see it increase after childbirth. The same is seen after tapping ascites, particularly if the patients sit up immediately afterward, undertake some violent exertion, or fail to take sufficient care of themselves in other ways.

The most frequent form of dislocation downward is the so-called subvertical position; next in order of frequency is the vertical position. Pronounced loop or crescent forms are less frequently seen. Vertical position is produced by dislocation downward of the freely movable pyloric portion of the stomach. Vertical and subvertical dislocations are the rule in cases of corset-thorax in women. Here the liver is contracted and forced downward so that the pylorus is also forced downward and inward. It is this peculiar vertical position of the stomach that can easily lead to dilatation of the stomach. Kussmaul first called attention to this, and showed that this form of dislocation occurs so readily because a variety of other factors are active at the same time that lead to atony of the muscularis of the stomach: for instance, general weakness, frequent overloading of the stomach, insufficient bodily exercise, etc. If the abdominal walls are relaxed at the same time, the occurrence of ectasy is also favored.

A glance at Fig. 17, which is taken from the well-known work of Kussmaul on *Peristaltic Unrest of the Stomach,* will show that the expulsion of ingesta must be rendered more difficult whenever the stomach occupies a vertical position. Not only is the stomach situated in an abnormal place; not only does it occupy the whole left side of the abdomen, but it is also considerably changed in outline. The fundus is smaller than normal, and the pyloric region occupies a position low down in the abdomen, so that it is readily distended by the stomach-contents. At the same time the lesser curvature is indented near the pyloric portion of the stomach. In severe cases this indentation may constitute an acute angle. Notwithstanding these abnormal conditions, which are all unfavorable to the propulsion of ingesta, the food does not necessarily remain in the stomach for an abnormally long time, particularly in mild degrees of this form of dislocation. In some instances, however, atony and ectasy develop.

Cases as severe as the one pictured in Fig. 17 are rare. Mild degrees of vertical or subvertical dislocation, however, are quite frequently encountered (compare Plates 1 and 2). These illustrations, as well as the other ones on Plates 3 to 6, are copied from nature in cases in which the stomach was slightly inflated with carbonic acid gas. Plate 1 represents simple vertical dislocation; Plate 2 the same form of dislocation, and at the same time ectasy caused by stenosis from ulcer scars. Plates 3 and 4 represent other forms of gastroptosis. In the latter cases there is also ectasy.

In Plates 5 and 6 other gastric anomalies are depicted—so-called loop, crescent, or garland forms. Here the stomach is dislocated downward and at the same times assumes the shape of a loop or of a crescent.

Numerous other modifications may be seen in addition to these main types. We rarely encounter simple dislocation downward of the whole stomach. In general, as Meinert emphasized, the dislocation is irregular—that is, different parts of the organ are dislocated to a different degree. This explains the occurrence of so many different forms. The only cases in which we can speak of a dislocation of the whole stomach downward are those in which the diaphragm moves downward, and with it the cardia, the fundus, and the whole stomach. In individual cases, of course, the distribution of space within the abdominal cavity will determine the position the stomach occupies when it becomes dislocated.

The appearance of so many different forms of abnormal position of the stomach makes it very improbable that gastroptosis is due to some uniform cause. Glénard, it is true, attributes all these cases to primary weakness of the ligamentous apparatus of the right colic flexure. He claims that these ligaments become relaxed, and that later the transverse colon, and secondarily the stomach, becomes dislocated downward.

As a matter of fact, however, a dislocation downward of the right colic flexure is absent in the majority of cases, so that this seems to contradict Glénard's view. It is unnecessary to enter into a complete refutation of his hypothesis. The most satisfactory explanation for all the anomalies of position that we have mentioned above is to assume that they are caused by the efforts of the stomach to occupy a position that corresponds to the space at its disposal within the abdomen at any given time. The great variety of processes that may occur within the abdominal cavity may lead to an abnormal distribution of this space, and consequently to dislocation of the stomach.

I need not say very much in regard to the methods of diagnosing this condition. It is clear that anomalies in the position of the stomach are more frequently overlooked than recognized. In the majority of gastric diseases clinicians content themselves with analyzing the secretory powers of the stomach. Only a comparatively small number attach much importance to the examination of the motor powers, and a still smaller number determine the position of the pylorus and of the lesser curvature. All that is done, as a rule, is to determine the lower boundary of the stomach—that is, the position of the greater curvature.

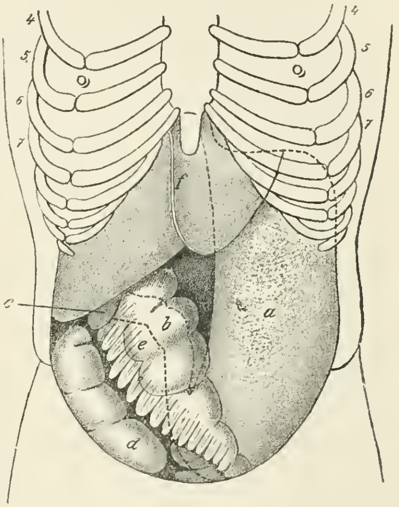


Fig. 17.—*A*, Stomach: those parts of the organ that were not exposed when the body was opened are designated by dotted lines: *B*, pylorus; *C*, gall-bladder; *D*, cecum; *E*, transverse colon, with the greater omentum rolled up and extending downward around the colon.

It is, as a rule, impossible to diagnose gastroptosis by the methods ordinarily employed at the bedside for determining the boundaries of the stomach—namely, palpation and percussion. Even Leube's method of percussing the gastric region with the patient in the erect position and after introducing water into the stomach with or without the sound is sufficient to determine only the lower boundary of the stomach, but gives us no information in regard to the position of the lesser curvature. In cases, of course, in which the stomach is very much dislocated downward, the position of the lesser curvature may occasionally be recognized in this way. A dislocation of the stomach downward can be diagnosed only if the position of the lesser curvature is determined, and the fact that the lower boundary of dulness is nearer the symphysis than normal does not necessarily prove that the patient is afflicted with gastroptosis.

In practice gastroptosis and ectasy are frequently confounded. Many clinicians commit the error of diagnosing ectasy whenever they find the greater curvature lower down than normal, but we know that gastroptosis alone may cause a lowering of this boundary. It is impossible to diagnose abnormal enlargement of the stomach, abnormal position of the stomach, or a combination of these two conditions without determining the exact location of the lesser curvature.

To judge from my personal experience, I should say that we possess only one method for determining at the same time the position of the greater and the lesser curvature, the total size, the position, and the outline of the stomach, and this method is artificial inflation. The stomach may be inflated with a double bellows or by administering an effervescent mixture; I prefer the latter method for the reasons chiefly that it can be executed at any time and does not require particular instruments nor apparatus, nor the aid of an assistant. In order to produce a distinct inflation of the stomach, however, large doses of the preparation must be given.

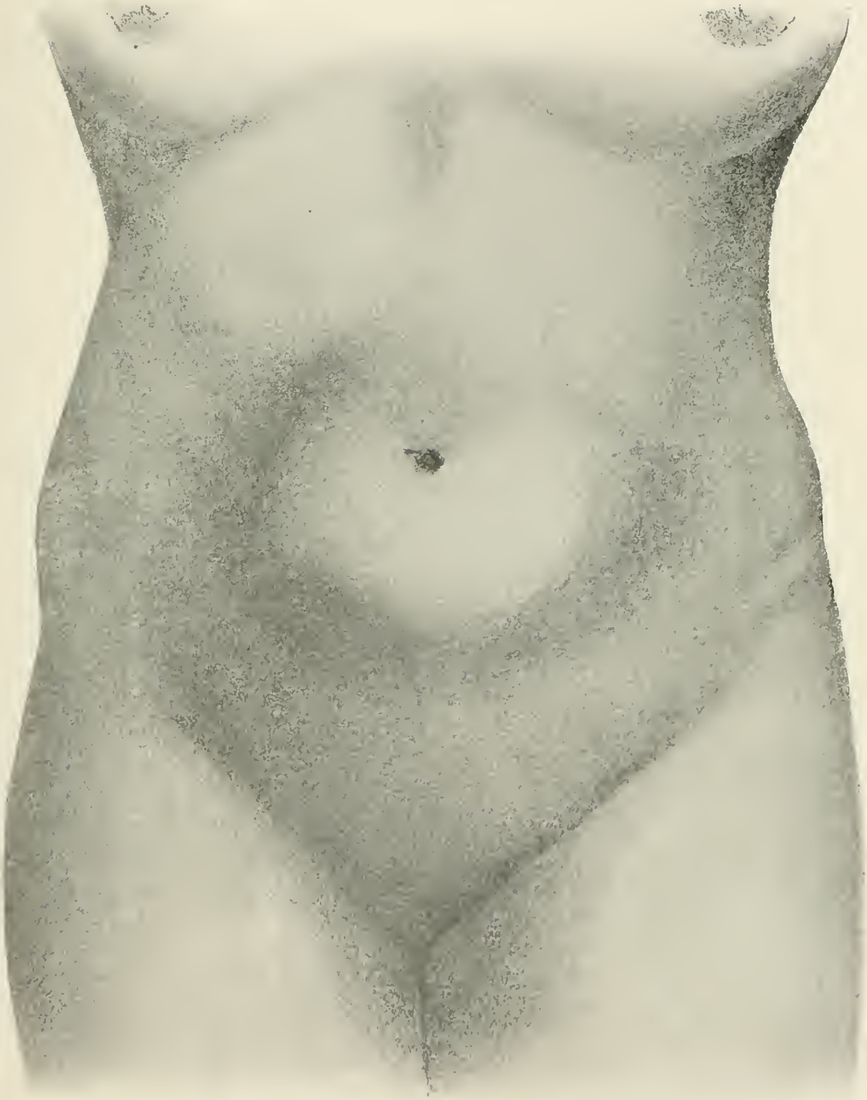
Insufflation of air with bellows may be employed advantageously in cases that are accustomed to the introduction of the stomach sound; in all other cases the administration of an effervescent powder is to be preferred. The latter method, if correctly performed, undoubtedly gives the most reliable results and enables us to outline distinctly the boundaries of the stomach. Some authors, it is true, doubt the efficacy of this method; I am inclined to believe that their scruples are due to the fact that they do not perform the method correctly or are not sufficiently experienced.

Another objection, however, has been formulated against the inflation of the stomach with carbonic-acid gas which is to a certain extent justified—namely, that the development of carbonic-acid gas in the stomach inflates the organ beyond its physiologic size. But assuming even that artificial inflation produces a picture that shows the stomach to be larger than normal, this, it appears to me, does not detract from the value of the method in diagnosing gastroptosis. For if the stomach is too much inflated, it may, it is true, become distended downward and anteriorly to an abnormal degree, but never upward, for in this direction the distention of the organ is prevented. In the case we are discussing the chief indication is to discover the position of the pylorus and of the lesser curvature, and this point can be determined with great certainty by inflating the stomach.

There is still another objection to the inflation of the stomach with an effervescent powder—namely, that this procedure frequently causes disagreeable sensations. I need hardly argue this point. All that it is desired to accomplish by inflation is to make the boundaries of the stomach visible and palpable for a brief moment. I always have a sound ready when I perform inflation in order to be able to evacuate the gas from the stomach as soon as symptoms of distress appear. I have performed artificial inflation daily for a long time, and have never seen any disadvantages or any serious symptoms from this method.

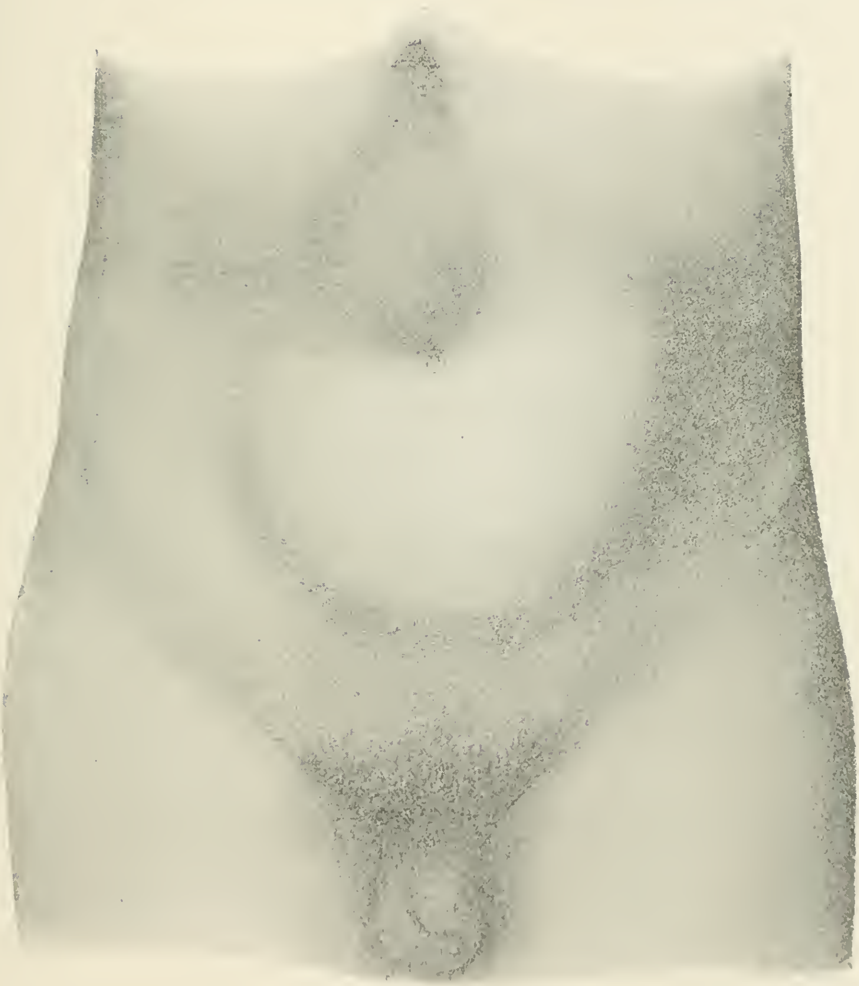
There are undoubtedly a number of cases in which simple inspection

PLATE 3.



GASTROPTOSIS IN A GIRL OF NINETEEN YEARS.

PLATE 4.



Gastroptosis with Ectasy in a Man of Forty-seven Years.

will reveal the fact that the stomach is situated low down in the abdomen. Here, it might appear, that artificial inflation would be superfluous, but it is always better not to content one's self with simple inspection, but always to control the diagnosis by inflating the stomach.

I will not enter into a discussion of the question whether or not transillumination of the stomach leads to the same goal with the same degree of certainty. Some authors claim this, but the results reported so far do not create the impression that this method is particularly suitable for determining the position of the stomach, and even if the results of transillumination were absolutely uniform and could be interpreted only in one way, the method would hardly be applicable in general practice because it is so complicated.

I have already mentioned that the different forms of dislocation downward of the stomach are more frequently seen in women and young girls than in men.

It is to be expected *a priori* that a change in the position of any one of the intra-abdominal organs would naturally lead to the dislocation of organs other than the stomach. For this reason contraction of the thorax by lacing dislocates not only the stomach, but also a number of other organs, particularly and most frequently the right kidney. A dislocation of the latter organ downward is seen particularly in corset-thorax. The explanation for this is simple enough. As soon as the thorax becomes contracted, the liver is rotated around its axis, the right lobe is depressed, and in this way pressure is exercised on the kidney. Other factors, like frequent and violent spasms of the diaphragm, rapid loss of adipose tissue, pregnancy, and confinement may all favor the development of this form of dislocation.

The degree of dislocation of the kidney varies. In some instances the lower portion of the kidney alone is palpable; in others the whole organ can readily be palpated and freely moved about. This is not the place to discuss the general symptom-complex of floating kidney. I will only call attention to the fact that floating kidney and gastrectasy are frequently found together, and that for a long time clinicians were inclined to see a connection between the two lesions. Some investigators claim that floating kidney is the primary event and gastrectasy the second; others assume the contrary. Nowadays we know that in many of the cases that are called ectasy we are not really dealing with ectasy of the stomach, but with gastroptosis, and that in others again there is a combination of gastroptosis and ectasy. Gastroptosis and floating kidney are not causally related to each other, but both are the effect of a third cause—namely, of some factor that causes a dislocation of the abdominal viscera, in general, downward.

The liver, too, is frequently found dislocated. Only in rare instances do we find the whole organ pushed downward. [Views as to the presence of floating liver have undergone a change, and it is now known that the liver, like the other viscera, may be displaced as a whole, and that it may move about quite freely in the abdominal cavity. To Einhorn is due the credit of pointing out the frequency with which this exists, as well as the methods to be employed in diagnosis.[1]—Ed.] This is seen only in cases in which the diaphragm is lower than normal. As a rule, the outline of the organ is changed; usually the liver is rotated in such a manner that the right lobe is pushed downward while the left one remains in its normal place. The liver also frequently shows the characteristic change of form that is known as corset-liver and that is caused by lacing.

Another organ that is frequently found dislocated is the colon, particularly its transverse portion. The dislocation of the colon can easily be diagnosed by inflating the bowels with air from the rectum, using a double bellows, as in the case of the stomach, or, as Boas has suggested, allowing water to flow into the rectum and determining the position of the large intestine by the location of succussion sounds. When the intestine is dislocated in this way, it is also frequently atonic.

I cannot enter into a discussion of all the other forms of dislocation of abdominal organs that are found together with gastroptosis.

We have already discussed dislocations *per se* and the methods we possess for diagnosing the different forms. It remains to describe the subjective and other symptoms caused by gastroptosis. We will see presently that there is a great diversity of opinion in this field.

Kussmaul, who was the first to call attention to these postural anomalies of the stomach, emphasized the fact that this condition is frequently complicated by a perversion of the motor powers of the stomach. Glénard, on the other hand, considered gastroptosis to be the anatomic basis of the protean symptom-complex that is grouped under the generic name of nervous disorders of the stomach.

The majority of modern authors are inclined to attribute an important rôle to these anomalies of position in the genesis of nervous and neurasthenic symptoms in general. Still others, among them Stiller, consider gastroptosis in its totality to be the effect of some congenital predisposition. Stiller claims to have seen most marked cases of enteroptotic neurasthenia in which the tenth rib was as freely movable as the eleventh and the twelfth. He considers this "costa fluctuans decima" to be a true neurasthenic or enteroptotic stigma.

It would lead us too far were we to discuss the question of enteroptosis in this place and its relations to nervous disorders, neurasthenia, and hysteria in general. In this section we must limit ourselves to a description of the anomalies in the position of the stomach and the direct consequences of these anomalies.

It is established that persons who are suffering from gastroptosis and other anomalies in the position of the stomach are frequently sufferers from dyspeptic disturbances and a variety of nervous, hysteric, and neurasthenic symptoms. All this, however, does not necessarily justify us in deducing any interrelationship between postural anomalies of the stomach and the above-named symptoms. Dyspeptic disturbances, above all, can hardly be regarded as nervous on these grounds; even in those cases in which other nervous and neurasthenic symptoms appear together with gastroptosis we are not justified in considering them *a priori* as the direct results of the abnormal position of the stomach. Nervous symptoms of this kind are frequently seen in cases in which the stomach is in its normal place, and, inversely, we find pronounced gastroptosis without any nervous disorders. I have seen gastroptosis in cases in which nervous symptoms, and, in fact, any symptom whatever, even stomach symptoms, were absent. Gastroptosis was discovered by chance in these cases when the attempt was made to determine the exact position of the stomach. When we consider how frequently gastroptosis is seen in the female sex (Meinert found the stomach in an abnormal position in more than 90 per cent. of his gynecologic patients), we need not be surprised that nervous dyspepsia and gastroptosis are found together, for both conditions are very common, particularly in women.

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[1] *Med. Record*, September 16, 1899.

From this point of view the investigations of Bial in men seem particularly interesting. It was to be expected *a priori* that nervous symptoms would be seen together with malposition of the stomach in men as well as in women, provided that the two disease-pictures were really related. Bial's investigations, however, showed that subjective symptoms of all kinds were absent in one-half of the men who suffered from gastroptosis. My own experience teaches me that the same applies to women.

Malposition of the stomach and certain nervous and hysteric symptoms are certainly frequently found together, but gastroptosis does not produce the nervous symptoms; there must be some other central condition, some particular nervous predisposition, that leads to the development of these symptoms. If this were not the case, the nervous system would never react by symptoms of this kind. This is the only explanation for the fact that nervous symptoms are occasionally seen in gastroptosis, but are absent in many other cases.

At the same time I do not wish to deny that gastroptosis *per se* may and frequently does lead to definite disturbance. Among these might be mentioned a variety of dyspeptic symptoms that consist either in perversions of motility, of secretion, or of the sensibility of the mucosa of the stomach. Wherever we find dyspeptic symptoms together with gastroptosis, we should attempt to determine whether these symptoms are due to some perversion of motility or of secretion, or whether they are purely sensory in character.

It is natural that a dislocation of the stomach downward, particularly if the organ assumes a vertical position or a looped shape, renders propulsion of the ingesta more difficult, especially when the patient is sitting or standing. No doubt the motor powers of the stomach are greatly overtaxed in these cases. There are, however, a large number of cases of gastroptosis in which this excess of work is not very great, in which the stomach is easily capable of performing the additional amount of labor, and in which the ingesta are propelled into the intestine within the normal time. As long as these patients live rationally and do not overtax the powers of their stomach, they suffer no distress; as soon as excessive work is thrown on the stomach, the organ becomes insufficient, the time of digestion is prolonged, and the patients begin to complain of a variety of symptoms that result therefrom, notably a feeling of pressure, fulness, and tension and of belching some time after eating. In many cases of this kind a repetition of indiscretions in diet may lead to dilatation of the stomach—to true ectasy.

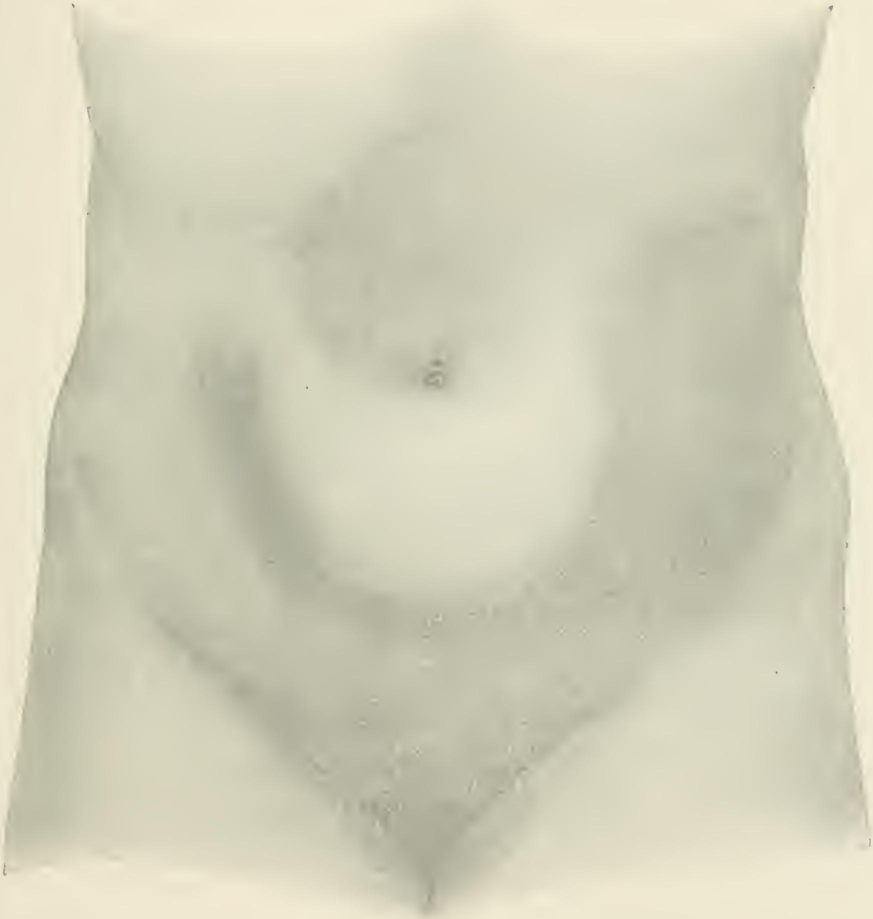
Kussmaul, several years ago, called attention to the fact that a mechanical obstruction to the exit of the food from the stomach exists in cases in which the stomach is dilated and dislocated downward, and that this obstruction is due to a bending of the superior duodenic flexure. An accident of this kind may occur suddenly, and very violent symptoms,—spasmodic pain, vomiting, etc.,—supervene. Cases of this kind, however, are rare.

Patients in whom the stomach is dislocated downward, particularly women, frequently complain of rumbling in the intestine, especially on the left side. As soon as they take a deep breath or walk fast or speak rapidly they are annoyed by this continuous rumbling. If the cases are more carefully investigated, the patients will usually state that the rumbling does not occur in the night—that it stops as soon as they remove their corsets, and that it never occurs when the organ is empty, but only after eating. I think Fleiner has found the correct explanation for this phenomenon when he suggests that these rumbling sounds are stenotic sounds caused by the compression of the abdomen by the clothing, chiefly the corsets.

Gastroptosis may occasionally lead to motor disturbances of the stomach. It may render the propulsion of ingesta from the stomach into the intestine more difficult. Less frequently, probably, it may lead to perversions of the chemism of the stomach. Disturbances of the latter kind are, it is true, occasionally found in gastroptosis, but they are probably not a direct result of the dislocation of the stomach.

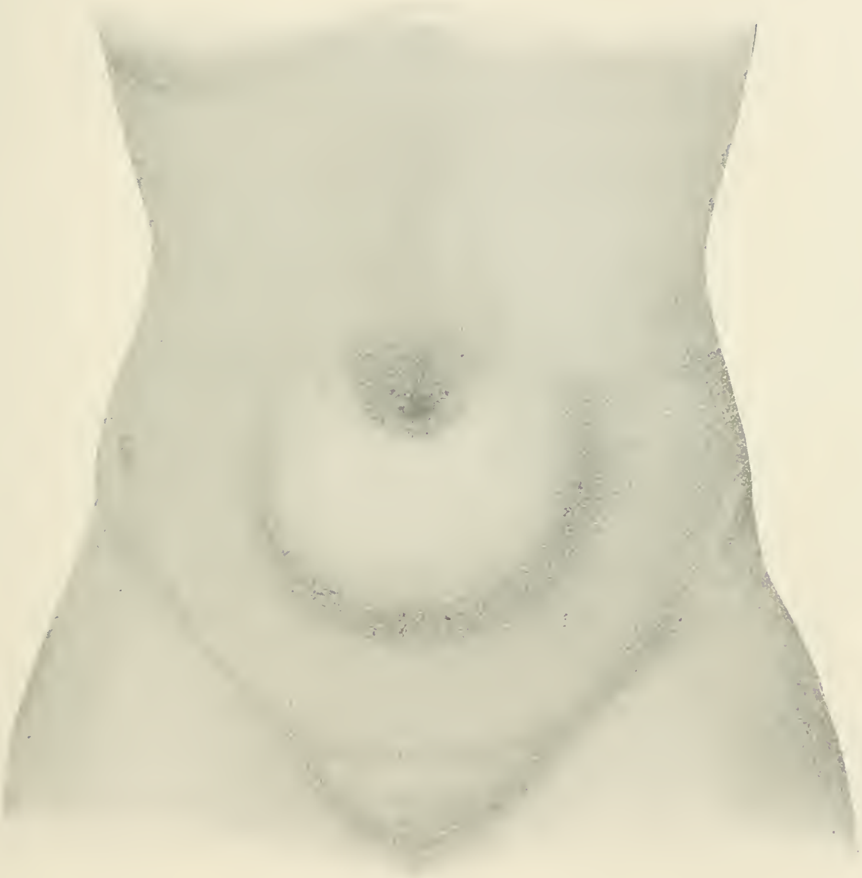
We have already mentioned that in severe cases of gastroptosis there may be motor disturbances that finally lead to gastrectasy. When this occurs, abnormal decomposition processes, fermentation with the development of gas, etc., may develop. If the ingesta undergo stagnation and fermentation and putrefaction develop as a result, the mucous lining of the stomach may become irritated, and in this way secondary perversions of the secretion result. This order of events is, however, rare. In those cases in which perversions of secretion are found together with gastroptosis, the former are, as a rule, due to some other cause; they are merely complications of gastroptosis which may occur in this disease as in any other disease of the stomach. There probably is no causal relationship between the two. In gastroptosis we may find either increased or more or less decreased secretion of gastric juice. In chlorotic cases we frequently find hyperchlorhydria, and at the same time we often see malposition of the stomach in such cases. Hyperchlorhydria and gastroptosis, however, are not dependent upon each other: they are merely concomitant conditions in chlorosis.

PLATE 5.



Gastroptosis with Crescent Form in a Woman of Thirty-one Years.

PLATE 6.



Gastroptosis with Crescent Form in a Woman of Thirty Years.

Many authors are inclined to consider the dyspeptic symptoms that are seen in gastroptosis, particularly if these are characterized by perversions of secretion (either in the sense of an increase or a decrease in the flow of gastric juice), as of nervous origin. This seems hardly justifiable. In many cases, if we will only examine carefully, the cause of these disturbances will usually be found, so that it is altogether unnecessary to seek refuge in the convenient hypothesis of nervous secretory perversions.

Certain disturbances in the sensibility of the stomach may also be caused by malposition of the organ. Gastroptosis that is caused by adhesions may naturally cause distress if the stomach becomes distended and overfilled. We are hardly justified, however, in considering the pain that is felt under these conditions to be a symptom of a purely sensory neurosis.

A variety of nervous disorders is seen in women with dislocated stomach, more frequently, in fact, in women than in men. Fleiner is undoubtedly correct when he states that changes in the form and the position of the stomach may lead to the same variety of nervous disturbances as changes in the position of the uterus. We are hardly justified in saying that a healthy human being becomes hysteric as soon as there is anteflexion, nor are we justified in claiming that malposition of the stomach causes neurasthenic or hysteric symptoms in any subject unless there be some central predisposition for the development of these states. Malposition of the stomach may cause local symptoms, and, as a matter of fact, does produce such symptoms in a small number of cases. It may also lead to certain disturbances in the motor sphere of the stomach, and can even secondarily lead to gastrectasy, and in this way to perversions of gastric secretion; finally, under certain conditions, it may lead to sensory disturbances. It is even conceivable that inflammatory adhesions may finally lead to the formation of an ulcer at some poorly nourished spot of the stomach. All other sequelæ to malposition of the organ are indirect and presuppose some particular predisposition—viz., either a neurasthenic or a hysteric taint.

[The stomach sometimes becomes twisted upon its axis, producing a state that may be called volvulus of the stomach, an instance of which has been reported by Wiesinger. Beck[1] also has had 2 cases in which he confirmed his diagnosis by operation, resulting in the complete cure of the patients.—Ed.]

Therapy.—It would be an easy matter to prevent the development of malposition and of changes in the form of the stomach. The most sensible prophylactic measure would be to reform our clothing, and particularly to combat the wearing of corsets. If women would adopt some other garb that did not force them to lace around the waist, gastroptosis would not be so frequent. Corsets that compress the thorax very much should, under all circumstances, be forbidden. Instead of wearing corsets, a so-called reform corset with shoulder-straps and buttons for attaching the skirts may be recommended.

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[1] *Centralbl. f. Chir.*, September 3, 1898.

During the lying-in period more attention should be paid to bandaging the abdomen, so that it may regain its former shape. Many women after delivery attempt to reduce the abnormal distention of the abdomen by lacing; they attempt to reëstablish their former waist measure. Lacing, however, forces the whole abdominal contents downward. It would be much better if the abdomen were tightly bandaged immediately after delivery and in this way the abdominal muscles forced to contract uniformly. At the same time meteorism should be treated and care should be taken that the stools are carefully regulated. The patients should not be allowed to get up before the tone of the abdominal muscles is completely restored.

Treatment proper of dislocation of the stomach is purely mechanical. It is frequently found that mild cases of gastroptosis are cured if the patient is forced by some intercurrent disease to lie in a horizontal position for several weeks or months. Pregnancy occasionally pushes the gastroptotic stomach upward. As a rule, however, the organ drops back into an abnormal position as soon as the child is born.

Rest-cures that force the patient to lie still sometimes act favorably, owing to the fact that the patient occupies a horizontal position so long, and that the abundant amount of nourishment that is administered leads to the deposit of a large amount of fat. Patients with gastroptosis should be instructed to lie down as much as possible, particularly after each meal. They should remain in the horizontal position, should relieve all pressure from clothing, untie all skirt-bands, etc.

The diet should be digestible, and should be carefully adapted to the secretory powers of the stomach. At the same time it should be so constituted that it is readily liquefied and can be propelled into the intestine in a short time. It is impossible to formulate any general rules in regard to the composition of the diet in cases of gastroptosis and of other malpositions of the stomach. If hyperchlorhydria exists, the diet will have to be different from that used if there is reduced secretion of gastric juice, and it will have to be planned on other lines if ectasy is present. The same dietary regulations apply here as in the case of all these different diseases. Two rules, however, should always be observed—namely, first, never to give much food at one sitting; second, to instruct the patient to occupy a horizontal position for some time after each meal.

In cases in which it is desired to improve the nutrition of the patient a milk-cure may occasionally be indicated—only in those instances, however, in which there is no objection to the introduction of large quantities of fluid.

Lavage is not called for in the treatment of malpositions of the stomach nor in dislocation of the stomach downward. Only if atony and ectasy appear may lavage be instituted. We refer to the sections on Atony and Ectasy for the details of this treatment and the various indications for its employment. What has been said of lavage applies to massage, electricity, and the administration of strychnin.

Massage of the stomach is without value in the treatment of dislocation of that organ; the stomach must be fixed and held in position by bandages. The form of the bandages will have to be adapted to each individual case; they will have to be arranged differently if the abdominal walls are flaccid; if there is diastasis of the recti muscles; if the abdomen is pendulous, or, on the other hand, if the tone of the abdominal muscles is normal. To judge from my personal experience, I should say that no one form of binder or bandage fits all cases. Even in cases of simple gastroptosis all pressure should be brought to bear on the symphysis and should act from below backward and upward. Occasionally a well-fitting rubber bandage will suffice for all purposes; in other cases some special apparatus must be constructed. Landau, Bardenheuer, Rosenheim, have described some useful forms of bandages. Wherever much pressure is brought to bear, a suitable bolster or pad should be inserted.

I need hardly emphasize the fact that cases of this character should not wear corsets of any kind. Instead, they may wear the reform corset that we have described above, with shoulder-straps and buttons.

In cases of gastroptosis and enteroptosis the stools should be carefully regulated. This can be done either by dietetic rules or by irrigation. If there is atony of the colon, the abdomen may be massaged; if possible, laxatives proper should be avoided.

If the abdominal walls are very much relaxed, massage of the abdomen, douches, particularly the Scottish douche, electricity, and cold rubs may be employed in addition to bandages.

Certain anomalies in the position and certain changes in the form of the organ can be corrected only by surgical means. If the stomach is dislocated by peritonitic adhesions or by scar tissue, the latter must be removed by surgical measures. The same operations can be performed as in hour-glass contraction of the stomach. There are a number of cases on record in which the latter condition was cured by surgical means.

Many forms are incurable. If the diaphragm is situated in an abnormally high position as a result of contraction of the lung following pleuritic exudates, this condition cannot be cured. The rare cases of dislocation of the stomach into the thoracic cavity, in certain defects of the diaphragm, in diaphragmatic hernia, also belong to this group.

CATARRH OF THE STOMACH, GASTRITIS, CATARRHUS GASTRICUS.

LITERATURE ON ACUTE AND CHRONIC GASTRITIS.

The following summary of the literature includes only the more important modern investigations on acute and chronic gastritis. For the old literature up to 1878 I refer to Leube's "Diseases of the Stomach" in *von Ziemssen's Handbook of Special Pathology and Therapy*. In this summary the literature of atrophy of the gastric mucosa and of phlegmonous gastritis is omitted. This will be found in the special chapters on these diseases.

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Compare also the well-known text- and hand-books and the monographs on diseases of the stomach by Boas, Bouveret, Ewald, etc.

Introductory Remarks.—Acute and chronic catarrh of the stomach are encountered with extraordinary frequency in practice. It would appear from this that the diagnosis of these conditions is very easy; as a matter of fact, however, it is quite difficult. The average practitioner apparently does not analyze the symptoms that lead him to make the diagnosis of acute, and more particularly of chronic, gastritis with sufficient care; if we look carefully, we will find that in the majority of cases we are dealing with a varied array of dyspeptic symptoms that are seen in many different diseases of the stomach. Again, many are in the habit of diagnosing gastritis by exclusion—*i. e.*, from negative evidence, from the absence of symptoms indicative of ulcerative or malignant processes, etc. If the latter can be excluded, most physicians feel justified in assuming that the dyspeptic symptoms are caused by gastric catarrh. If this line of reasoning is pursued, many errors are naturally committed. It would be better if physicians would consider positive evidence alone, and would diagnose gastritis or gastric catarrh only in those cases in which a sufficient number of positive symptoms can be discovered.

Cases are frequently seen that complain of certain dyspeptic symptoms,—a feeling of pressure and fulness after eating, acid eructations, heartburn, and similar disturbances,—and that may have undergone treatment at the hands of many different physicians for “chronic catarrh of the stomach.” An examination of these patients usually fails to reveal any typical symptoms ; all that is generally found is diffuse sensitiveness to pressure over the region of the stomach. If the stomach-contents is analyzed after a test-breakfast or after a test-meal, certain changes of the food residue will be found, in a small proportion of cases, which correspond to chronic catarrh of the stomach.

In a large proportion of the cases, however, that come to the physician with such dyspeptic symptoms, aspiration of the stomach-contents will reveal nothing more than an increase of the hydrochloric acid—namely, *hyperaciditas hydrochlorica.* Are we justified in calling these cases chronic gastritis ? I think not. The subjective symptoms and the whole disease-picture may resemble chronic gastritis greatly and still not justify us in making this diagnosis. We may speak of dyspepsia with hyperacidity, or we may speak of hyperacidity alone, or of acid dyspepsia, but we cannot make the diagnosis gastritis from the symptoms we find. I do not wish to deny, of course, that hyperacidity may occur together with gastritis ; I will refer to this question in detail further on. In this place I am merely discussing the cases of pure hyperacidity that we occasionally encounter. Whenever we speak of catarrh or gastritis, we always mean that the epithelial layer of the mucous membrane of the stomach is diseased and that the secretion of the stomach is abnormal. If the disease progresses further, we see parenchymatous or interstitial inflammations of the stomach-wall. In the case of the stomach mucosa matters are more complicated than in any other mucous membrane, for the stomach does not really possess a mucous lining or a mucosa in the ordinary sense of the word, and in the case of the stomach it is almost impossible to draw a distinct line between catarrh and parenchymatous inflammation. The superficial epithelium of the gastric mucosa is, at the same time, the epithelium of the tubules, and the former epithelium is in direct continuity with the epithelium of the gastric glands. In any catarrh of the stomach, therefore, the gastric glands are always to a certain extent involved, and still we may hardly be justified in speaking of a catarrh of these glands. In the case of the stomach, therefore, we may say that we are always dealing with an inflammatory process—a gastritis proper. This gastritis may assume different degrees of severity ; it may be limited to the superficial layers of the mucous membrane, may extend down to the glandular parenchyma proper, or may involve the interstitial tissues.

We see, therefore, that it is impossible to formulate any distinct difference between catarrhal and parenchymatous inflammation of the stomach. Neither pathologic-anatomic findings nor clinical symptoms allow us to do this. Hoffmann, in order to obviate this difficulty, advises clinicians to drop the term catarrh of the stomach for those cases in which superficial inflammation involves the functionating cells of the stomach, and advises calling the milder degrees of this form of inflammation desquamative inflammation. Ewald also recommends dropping the term catarrh of the stomach altogether, and supports his point of view by a number of arguments.

What we are in the habit of calling catarrh of the stomach is usually the parenchymatous form of inflammation. This applies still more to the chronic than to the acute forms. The longer the epithelium is diseased, the more may we expect to see the inflammation extend to the parenchyma proper. In still other cases the gastritic process is interstitial from the beginning. Clinically, we are unable to distinguish between parenchymatous, interstitial, and desquamative forms, at least not in mild degrees of these different inflammations. In general, all these different forms are grouped under the name gastritis. The degree of inflammation varies according to the duration of the affection, its primary cause, and other factors.

Gastritis, according to its duration and course, must be divided into an acute and a chronic form. This subdivision is analogous to the one we have formulated in acute and chronic catarrh of the stomach.

Different authors disagree in regard to the most practical manner of continuing this classification. Ewald[1] distinguishes an acute, simple chronic, and a mucous gastritis, and also a fourth form, which he calls anadeny, or atrophy of the stomach. A separate form of gastritis, finally, is the phlegmonous or suppurative form.

Other authors speak of a toxic or an infectious gastritis. Penzoldt[2] advises dividing gastritis into a mild and a severe form, and advises against suddenly replacing the anatomic method of classification by the etiologic one. This, he argues, is done if we speak of a toxic or an infectious form of gastritis. I agree with Penzoldt in this objection, for every gastritis is toxic in the broad sense of the word. At the same time the above method of subdivision is justified to a certain extent from a purely clinical point of view, chiefly because the general symptom-complex, the degree of gastritis, and the course of the disease may vary in different respects according to their origin. Gastritis, for instance, which is caused by some error of diet, is, as a rule, mild and of short duration ; gastritis, on the other hand, which is caused by swallowing acids or caustic alkalis usually produces much more violent symptoms. Between these severe and mild forms many intermediate stages can be distinguished. Clinically, therefore, we are justified in distinguishing between mild and severe forms of gastritis. From a practical point of view, an etiologic classification of the different forms of gastritis may be justified ; but I do not think it imperative to give up the anatomic method of classification altogether in favor of the etiologic one.

(*a*) SIMPLE ACUTE GASTRITIS; ACUTE CATARRH OF THE STOMACH.

Etiology.—Simple acute gastritis—acute catarrh of the stomach—is a disease that is encountered daily. We designate by these terms an acute inflammation of the superficial layers of the gastric mucosa characterized by an increased secretion of mucus and desquamation of the epithelial cells. The epithelium of the rennet glands is, as a rule, more or less involved.

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[1] *Real-Encyklopädie*, vol. xxiv.

[2] *Handb. d. spec. Therap. innerer Krankh.*, vol. iv.

This acute gastritis may either be a primary and independent disease, or it may be a secondary condition following or accompanying other diseases of the stomach. In the majority of cases some direct irritant, mechanical, chemical, or thermic, etc., injures the mucous membrane of the stomach and causes an acute inflammation.

The mucosa of the stomach may be irritated and inflamed by food or drink that is too hot or too cold. Thermic irritation will produce different effects if the stomach is empty or if it is filled with food; in the latter case the effect of the thermic irritant will be less severe than if the hot or the cold article of food enters the stomach and comes in immediate contact with the mucous lining. The same applies to very irritating articles of food, strong spices, drugs, and poisons that are local irritants and may produce acute gastritis. Too large quantities of food, particularly of coarse material, may not be sufficiently digested by the gastric secretion, and consequently remain in the stomach so long that they cause acute atony, irritation of the mucous lining, and gastritis.

It might be argued that many of these noxious agencies do not necessarily always produce gastritis, and that they frequently enter the stomach without producing gastritis. This objection is certainly valid. We must never forget, however, that the stomach is an organ whose capacity and whose sensitiveness vary in different individuals; and, again, the stomach is an organ that may gradually become accustomed to an abnormal amount of labor. Certain individuals can impose an enormous amount of excessive work on the stomach; they can eat hot and cold food in rapid succession, can digest the most undigestible material, and still suffer no distress. On the other hand, there are many persons who are sufferers from what may be called a weak stomach. In these subjects the slightest deviation from the normal method of life, the least excess, immediately leads to dyspepsia and gastritis. We can speak of a particular predisposition to certain diseases in the case of the stomach as in the case of any other organ. Such a predisposition, it appears, is frequently hereditary. Many patients of this character will state that their father or their mother also had a weak stomach, and that their brothers and sisters are afflicted in the same way. In these cases, of course, it must be decided how much of this is due to heredity and how much to abuses that the stomach had to suffer when the subjects were very young. We know that the stomach, like the muscles of the heart, can be educated to a certain standard of efficiency. This education, however, must always be begun during childhood. We frequently find that in families where one or both of the parents are afflicted with a so-called weak stomach the children are accustomed to a uniform and monotonous diet from early childhood up; as a result, the stomach is unable to accomplish any unaccustomed work. As soon as it becomes slightly irritated, gastritis develops. I think that Hoffmann goes a little too far when he says, in his excellent work on "Therapy," that "every person has a stomach that is as good as he deserves." This dictum, however, certainly applies to the cases we have described above.

The lower classes of our city population and our peasant population usually have stronger stomachs than our city-reared children. This is due to the fact that the former are forced to eat a more voluminous and more indigestible diet, containing more residue, than the latter, consequently the stomach becomes accustomed to mastering larger quantities of coarser food. Even in every-day life the influence of habit is manifest; certain patients, for instance, who live for a number of weeks on a liquid diet frequently suffer from a variety of gastric symptoms, as pressure, belching, and other signs of acute dyspepsia, as soon as they eat food of greater consistence for the first time, or as soon as they attempt to eat even a small quantity of solid food that they could have mastered very well before they were sick. It remains to be decided, of course, in many of these cases, whether the symptoms were due to acute gastritis or to some simple functional disorder of the stomach.

Another prolific cause of acute gastritis is the ingestion of spoiled food and drink. Particularly in summer do we encounter this form of gastritis; it may even be endemic or epidemic. Certain products of putrefaction or micro-organisms usually play an important rôle in the causation of this form of gastritis. Micro-organisms, if they are present in the food, can undoubtedly cause gastritis, or, better, gastro-enteritis. As a rule, the gastric symptoms are not so important and not so conspicuous as the intestinal symptoms. Gaffky[1] has reported an observation that he made in 3 persons who were infected from raw milk that came from a cow suffering from hemorrhagic enteritis. I saw two of these cases myself, and could determine that they presented the picture of a severe infectious disease. The cause of the disease was found to be a very virulent form of the Bacterium coli communis.

Gaffky[2] has also shown that many forms of meat- and sausage-poisoning that present the picture of acute infectious gastro-enteritis are due to the presence of certain micro-organisms.

Primary mycoses of the stomach have repeatedly been observed. A number of localized mycotic lesions of the stomach have also been reported that were not primary, but merely manifestations of a general infection. We are unable so far to delineate a typical clinical picture of *gastritis mycotica*. Symptoms of gastric ulcer have frequently been seen in suppurative processes of different parts of the body, in dysentery, in lymphangitis, in abscess, and in chronic glanders. It is possible that these cases were true mycoses of the stomach, but the diagnosis could hardly ever be made with certainty. It has also been found that diphtheritic processes may, under certain conditions, extend to the mucous lining of the stomach. Anthrax bacilli (Birch-Hirschfeld) and numerous other micro-organisms may also lead to gastritis, even to the formation of ulcer. In all these so-called infectious forms we may speak of infectious gastritis if we wish to attach the chief significance to the etiologic factors of the lesion.

Schizomycetes may also develop in the stomach, particularly in children and old people. Rosenheim has further reported a case in which thrush caused violent gastric symptoms. In this case it found particularly favorable conditions for its development.

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[1] *Deutsch. med. Wochenschr.*, 1892, No. 14.

[2] *Arbeiten aus dem. k. Gesundheitsamte*, vol. iv.

The patient was a woman of sixty years who had been a sufferer from gout for many years, and was consequently obliged to live in bed. This old lady had been suffering for many months from loss of appetite, pressure over the stomach, and nausea, without being able to give any good reason for the appearance of these gastric symptoms. The stomach symptoms continued to increase in severity, the patient became very much reduced, and finally was unable to take even milk, which, up to a short time before, she had been able to drink without any difficulty. On examination, the tongue was found to be very much coated, there was a mild degree of stomatitis, and slight sensitiveness to pressure over the stomach. One day a very violent attack of vomiting occurred immediately after taking a little food. In addition to remnants of food and tough mucus, shreds of a doughy material were found in the vomit that formed either small lumps or larger pieces of membrane as large as an adult hand. These shreds were grayish or brownish-gray in color, and consisted of a veil-like arrangement of thrush that inclosed numerous leukocytes and free cell-nuclei. As soon as this mass had been raised and gotten rid of, the gastritic symptoms disappeared and the patient rapidly improved.

A rare form of gastritis that has occasionally been seen is the parasitic form. Cases have been reported in which maggots, larvæ of flies, rain-worms, and diptera larvæ entered the stomach with the food and caused gastritis ; and other cases where ascarides, tenia, and even oxyuris entered the stomach from the intestine and also produced gastritis. In these cases the symptoms of the disease disappeared as soon as these parasites were gotten rid of.

Toxic gastritis is frequently described in a special group ; under this heading are included those forms of inflammation of the stomach that are caused by mineral acids, by caustic alkalis, by certain metallic salts, by alcohol, arsenic, phosphorus, corrosive sublimate, potassium chlorate, and similar substances. If the superficial layers of the mucosa are involved alone, we speak of the mild form of gastritis ; if deeper layers of the stomach-wall are destroyed or inflamed, we speak of the severe form. The effect of these poisons, however, not only becomes manifest in the gastric mucosa, but may extend to other organs. It is impossible to discuss all these different forms of intoxication and poisoning in this place, as it would lead us much too far. We refer our readers to the section on Poisoning.

The effect of cold and of high degrees of heat on the surface of the body has also been made responsible for the development of acute gastritis. Erichsen[1] found pronounced congestion of the stomach and intestine in 12 of 14 cases of serious burns that he examined within the first twenty-four hours after the accident. Among 22 cases that he examined between the second day and the second week he found congestion and inflammation of the stomach and intestine in 11 cases, and ulceration of the duodenum in 6.

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[1] *Science and Art of Surgery*, vol. i., p. 375.

We also see frequently gastritic symptoms as a secondary condition. Gastritis, it is true, is more frequently found in chronic than in acute diseases. We will speak, later on, of the relation between chronic diseases of the lungs, heart-lesions, chronic diseases of the liver and kidneys, a number of constitutional diseases like diabetes and leukemia, and their gastritic symptoms. In this place we will mention only the acute forms of dyspepsia and gastritis which are seen in the course of acute diseases. Much remains to be discovered in this field. A few interesting observations are, however, on record. Fenwick[1] found that in the majority of cases of acute parenchymatous nephritis there was, at the same time, acute tubular gastritis. Wilson-Fox[2] reports 12 cases of diseases of the kidneys, 9 of which were acute and 4 chronic. In all the acute cases there was also acute catarrh of the gastric glands. Fenwick also found pronounced changes in the epithelium of the gastric glands in scarlatina.

Many acute febrile diseases, like erysipelas, measles, variola, and pneumonia, are accompanied by catarrhal affections of the gastro-intestinal tract. This is usually discovered on autopsy. In the majority of cases symptoms that are characteristic of gastritis are usually absent during life. Beaumont[3] stated in his day that there may be widespread catarrhal inflammation of the gastric mucosa without any clinical symptoms.

It is well known that in acute febrile diseases gastric symptoms frequently develop. In acute gastric processes, of course, that are due to infection or to poisoning fever is secondary. On the other hand, fever *per se* may, under certain circumstances, exercise an influence on the functions of the stomach. In regard to this point, it is true, there is a great diversity of opinion. Manassein[4] was the first to show that the gastric juice of dogs with fever did not possess as great peptic powers as the gastric juice of healthy animals, and that the former required the addition of hydrochloric acid in order to digest the same quantity of albumin as normal gastric juice could digest without the addition of acid. In examining human subjects with fever Uffelmann[5] found that the hydrochloric acid of the gastric juice was reduced in some cases and in others was increased. Sassezki[6] also found that the quantity of hydrochloric acid varied in subjects with fever. He always, however, found an absence of hydrochloric acid if the fever patient suffered from dyspepsia. Hildebrand[7] performed a number of investigations in my clinic that make it probable that fever *per se* can exercise a certain influence on the production of hydrochloric acid. The investigations of Hildebrand extended only to cases of phthisis, so that his results do not apply *eo ipso* to all febrile diseases. In the cases that he examined a direct damaging influence of the fever on the secretion of gastric juice could be determined. He also succeeded in causing an increased secretion of hydrochloric acid, even to the extent of producing free hydrochloric acid in the stomach-contents, by administering large doses of antipyrin to cases of phthisis that had fever, and in whom free hydrochloric acid was absent. Free hydrochloric acid appeared as soon as the temperature dropped. Even though it should be found that fever can exercise a damaging influence on the secretion of gastric juice, or, better, that an elevation of temperature can exercise this effect, we will not be justified in assuming that any true gastritis can be due to fever alone. In none of our cases were there true gastritic symptoms, and the only demonstrable change in the stomach was the decreased production of gastric juice.

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[1] *Virchow's Arch.*, vol. cxviii., p. 349.

[2] *Diseases of the Stomach*, p. 124.

[3] *Exper. and Observ.*, Coombe's edition, p. 171.

[4] *Virchow's Arch.*, vol. lv.

[5] *Die Diät in acut fieberhaften Krank.*, 1877; *Deutsch. Arch. f. klin. Med.*, vol. xx.

[6] *St. Petersburg. med. Wochenschr.*, 1879.

[7] *Deutsch. med. Wochenschr.*, 1899, No. 15.

The Anatomic Changes Observed in Acute Gastritis.—We know very little of the finer anatomic changes seen in acute primary gastritis. This is due to the fact that patients with this disease rarely die. On the other hand, we must never forget that postmortem changes occur more rapidly in the stomach than in any other organ. Only when particular precautions are taken are we able to gain a true histologic picture of the conditions that existed in the stomach during life. In order to render a histologic postmortem examination of the stomach more easy, many animal experiments have been performed; the gastric changes observed in postmortem examination of subjects that died from acute febrile diseases have also been studied. However valuable investigations of this kind may be, the results obtained by this method cannot be directly applied to simple genuine gastritis.

The following macroscopic changes in the gastric mucosa are seen in acute gastritis. The mucous membrane is, as a rule, more or less reddened and swollen, either diffusely or in circumscribed areas, and is covered with tough, usually cloudy, mucus. Here and there small hemorrhages may be found.

Beaumont described changes of this kind in his celebrated patient, the Canadian with a gastric fistula. This patient at one time developed an acute catarrh of the stomach that lasted for a short time, and Beaumont examined the lining membrane of the stomach and found that the mucosa was slightly swollen, covered with tough mucus, and roughened, particularly in the region of the pylorus. The gastric juice had a slightly acid, neutral, or alkaline reaction, and the food remained in the stomach for several hours without being digested.

This observation of Beaumont alone shows us that in acute gastritis the secretion of the gastric juice is more or less reduced, and that, consequently, digestion is disturbed.

The finer changes that are seen in acute gastritis occur chiefly in the surface epithelium, the glandular epithelium, and the interstitial tissues. The superficial epithelial layer is partly loosened, shows mucoid degeneration, and is in a state of cloudy swelling. In the glandular epithelium no difference can be seen between the parietal and the peptic cells; all the cells are in a state of cloudy swelling and fatty degeneration, and all are very much contracted. The capillaries in the superficial layers of the mucosa are much dilated. In the interstitial tissues there is an abundant accumulation of round-cells. Acute gastritis in its totality, therefore, presents the picture of a more or less pronounced hyperemia of the mucosa, with desquamation of the epithelium and involvement of the glandular epithelia; also occasionally mild symptoms of irritation of the interstitial tissues.

Symptoms.—Simple acute gastritis may, of course, assume different degrees of severity. As a rule, the disease causes no fever; in a few cases a mild type of febrile disturbance may be observed. We distinguish, therefore, the febrile from the afebrile form of gastritis. These two forms, however, coincide in all essential points. As a rule, a series of subjective symptoms develop soon after the noxious agency that inflicts the damage to the stomach begins to act. There are loss of appetite, a feeling of discomfort, and pressure and fulness in the region of the stomach; in a short time nausea and belching develop. The gas that is raised is usually acid, bitter, and disagreeable to the taste. Vomiting frequently occurs, and malodorous, acid, disagreeable remnants of food that are either altogether undigested or only slightly digested, and are frequently in a state of fermentation, are evacuated. In many cases vomiting occurs very soon after the introduction of the disease-producing agency; in other cases some time elapses before vomiting occurs. In the latter instance remnants of food appear in the vomit that were introduced into the stomach hours before. Considerable quantities of mucus are also usually seen in the vomit. Patients frequently bring about vomiting artificially if it does not occur spontaneously.

The reaction of the vomit is usually acid, but the total acidity of the stomach-contents is usually reduced, and free hydrochloric acid is, as a rule, absent. Organic acids, as lactic acid, butyric acid, and acetic acid, can frequently be found. In rare cases the stomach-contents may be neutral. If vomiting is very severe and frequent, bile constituents are occasionally raised. Sometimes the vomit may consist of pure bile. If a test-breakfast is administered to these patients as soon as the first violent symptoms have abated, and if the stomach-contents are pumped out an hour afterward, the particles of bread that were eaten will be found in a coarse state and mixed with mucus. They look as if they had just been chewed and swallowed. The reaction of the stomach-contents is only slightly acid, and free hydrochloric acid is, as a rule, absent.

An objective examination of the patient reveals little that is characteristic; the tongue is usually coated with a thick, tough, grayish layer of mucus, and appears swollen. The imprint of the teeth can usually be distinctly seen on its edges. The tongue does not regain its normal red color until the condition of the patient begins to improve, the redness first appearing at the edges and on the point of the tongue. Patients complain of a bad, acid, or stale taste in the mouth; the appetite is altogether lost, and there may be absolute disgust for food. The thirst is usually increased.

The region of the stomach is usually somewhat distended and sensitive over its whole extent; it may be painful spontaneously or on pressure.

Other subjective symptoms are headache, vertigo, a feeling of lassitude, and weakness. The intensity of these symptoms, of course, varies in individual cases; the urine is scanty, dark, of a high specific gravity, and contains much urates; occasionally it contains a little indican. The pulse is usually accelerated and easily compressible. A retardation of the pulse-beat is rare, and is found only in the severe forms of acute gastritis. The course of the disease varies; in mild forms the patients feel very much relieved immediately after vomiting and all the symptoms rapidly disappear; in more severe forms vomiting occurs repeatedly and the patients complain of constant nausea, even though nothing is introduced into the stomach. If vomiting occurs under these conditions, nothing is raised but mucus and bile. In many cases the irritation of the stomach extends to the intestine, so that more or less severe attacks of diarrhea occur at the same time. A tendency to constipation is more frequently seen than diarrhea, particularly in the first days of the disease. If the duodenum is affected, icterus may even develop.

The different symptoms that we have described do not, of course, occur with the same intensity in all cases. In milder cases there is no vomiting at all, only a little nausea, a feeling of discomfort in the region of the stomach on pressure, and a lack of appetite. All these symptoms disappear in the course of a few days, provided the patient is careful in what he eats and does not overtax his stomach. The appetite soon returns, diuresis increases, and the urine grows light and clear. In more severe cases the symptoms continue for a longer time,—usually for a number of days,—then gradually subside in severity until a cure is effected gradually.

I have already mentioned that simple acute gastritis usually runs its course without fever; there are, however, certain cases that are accompanied by a rise of temperature. This is particularly the case in children and young subjects; here acute gastritis is complicated frequently by a mild or severe degree of fever. A chill rarely ushers in the disease, although the patients frequently complain of a feeling of coldness, followed by heat. The temperature rarely rises very high; in exceptional cases, however, it may reach 39° C. or even more. When this occurs, other symptoms appear: there are great lassitude, pain in the limbs, etc. Eruptions of herpes labialis are also occasionally observed.

The fever is usually of the remittent type; the remissions gradually occur at less frequent intervals, and in a short time the temperature drops back to normal. In other cases, again, the fever terminates by crisis. In general, however, acute gastritis is not complicated by fever. Whenever we see fever together with gastritis, we must always suspect that the gastritic symptoms are secondary to some other cause, and are only a symptom of some primary disease. It would be better if the term "febris gastrica" were omitted from our nomenclature, for cases that are designated in this way are usually simple acute gastritis, or they are some other disease, like typhoid or influenza, that is accompanied by stomach symptoms. In the former case the name "gastritis" is sufficient; in the latter, the condition should be designated by the name of the disease. It cannot be denied, of course, that it may be difficult to make a positive diagnosis in cases of this character, particularly during the first days of the disease.

Diagnosis.—Simple acute gastritis without fever is usually easy to diagnose. Acute gastric symptoms, it is true, do not justify us in diagnosing acute gastritis offhand; the vomit must be examined, or the stomach-contents must be removed and analyzed. If coarse and undigested particles of food are discovered, particularly remnants of a meal that was eaten several hours before, or if there is an abundant admixture of mucus, if there is more or less subacidity, and if organic acids are present, the diagnosis can, of course, be made, especially if the history of the case shows that the stomach was injured directly some time before by overeating or by the ingestion of indigestible food.

In many cases a variety of symptoms are seen in addition to gastric symptoms, namely, certain nervous symptoms, like headache, a sensation of fear, intercostal neuralgia, herpes zoster; there may also be dyspnea, and all these symptoms may be so prominent that the significance of the gastric symptoms may be underestimated. This is particularly the case in nervous individuals. If the history of these cases, the etiology, and the course of the disease are carefully investigated, the diagnosis should not, however, be difficult. In severe forms of toxic gastritis in which symptoms of general intoxication or of infection supervene, the diagnosis may be rendered particularly difficult.

Acute gastritis can hardly be confounded with gastric ulcer or with gall-stone colic. Ulceration of the stomach never produces symptoms that resemble acute gastritis unless there is acute circumscribed peritonitis. In gall-stone colic violent pain is the predominant feature, and this is never found in simple acute gastritis. The latter disease can never be confounded with acute peritonitis nor with exudative pleuritis on the left side and similar conditions, provided the patient is examined carefully. It also seems impossible to confuse acute afebrile gastritis with any other disease of the stomach. Chronic diseases of the stomach are excluded; and of the acute forms of gastric disorders, the only one that could possibly lead to error is nervous gastralgia accompanied by hyperacidity; even here, however, the differential diagnosis is easy. Attacks of nervous gastralgia are, as a rule, caused by some psychic excitement, mental overexertion, anger, etc. The most prominent symptom is pain that usually lasts for a few hours only. If vomiting occurs, the material raised consists of very acid food-particles, and never contains products of abnormal fermentation, mucus, etc. The only abnormality to be discovered is an increase in the hydrochloric acid. In subjects who are predisposed to this form of gastric attack a history of previous seizures can usually be elicited, for attacks of this character never occur singly, but are usually repeated.

Another form of acute gastritis may lead to error, and that is acute febrile gastritis. I am personally of the opinion that fever is rare in acute gastritis. If there is fever together with pronounced gastric symptoms, we should always be suspicious of some acute infectious disease like typhoid. The spleen should be examined carefully. It is a well-known fact that in practice many cases that are not fully understood are conveniently called "gastric fever," and that many obscure infections are probably included under this term. We should speak of febrile gastritis only in those cases in which dyspeptic symptoms appear together with fever, and in which the temperature drops to normal as soon as the dyspeptic symptoms disappear.

It is hardly necessary to say anything in regard to the prognosis. Simple acute gastritis always terminates favorably. There is a certain danger in recurrences; the stomach frequently is exposed to the same damaging influences, and if this occurs, acute gastritis may gradually develop into chronic gastritis, followed by secondary atony and other sequelæ.

Treatment.—Acute gastritis can undoubtedly be prevented in many cases; in other instances it appears that the patient could not possibly have predicted that his stomach would become disordered. Acute gastritis frequently follows the ingestion of spoiled or adulterated food; here the patients frequently cannot know that the food they are eating is harmful. The individual is hardly responsible for this; the blame must be attached to those authorities whose duty it is to supervise the food that is in the market, and to determine whether it is adulterated or spoiled. If patients, on the other hand, are in the habit of indulging in excesses; if they expose themselves to influences that they know will damage the stomach,—for instance, if they persist in eating food that is too hot or too cold or is indigestible,—they alone are, of course, to blame. It is the duty of the physician to prescribe a rational mode of life and a rational dietary for patients who are attacked by acute gastritis whenever they indulge in excesses of this kind. Advice of this character is, of course, rarely followed. In the case of children, who are particularly predisposed to gastritis, it is the duty of the parents and of the physician to see that the diet is selected carefully; overloading the stomach with sweets, fruit, cake, etc., is a daily occurrence in the life of a child. This easily leads to fermentative processes, so that the stomach becomes abnormally distended and may even grow ectatic. Children frequently are afflicted with gastritis from eating unripe or dirty fruit; fruit should always be cleansed and washed thoroughly before eating; parents should never allow their children to eat unpeeled pears, apples, and fruit of that kind. All these prophylactic measures are, of course, so self-evident that we need not enter into their discussion any further, even though these common-sense rules are violated daily.

In regard to treatment proper, I might say that it is hardly necessary in the majority of cases; in many instances acute gastritis is cured without any assistance, and cases of this kind usually run a very short course.

A restitution to normal may occur in two ways: the stomach may either get rid of the ingesta by vomiting, or the patient may lose his appetite and refrain from eating for some time. If the physician wishes to treat cases of this kind at all, he should treat them along these two lines.

In cases where the stomach cannot get rid of the ingesta by vomiting, or where the evacuation of spoiled food is incomplete, the stomach-contents should be removed artificially. This can be done either by emetics or by lavage; the latter method is the more rational of the two. All internal emetics, particularly tartar emetic, which is still in universal use, are bad, for the reason chiefly that they irritate the gastric mucosa and because some time elapses before vomiting occurs, and the patients feel very uncomfortable in the mean time.

Some persons find it exceedingly difficult to vomit; some patients can vomit if they experience the slightest nausea; others again can hardly perform the act of vomiting, even though they be exceedingly nauseated, and these latter cases are frequently severely prostrated after the administration of an emetic. If it is desired to produce emesis by drugs, apomorphin should be administered subcutaneously, for this remedy possesses the advantage of acting more rapidly for a shorter time and with greater certainty. At the same time it does not irritate the gastric mucosa.

It is always more rational, however, to evacuate the stomach-contents by means of the stomach-tube in all cases in which the stomach is unable to get rid of its contents spontaneously. Physicians themselves are responsible for the fact that this method is not universally adopted in practice. If the stomach-tube is introduced by an experienced hand, no disagreeable symptoms are complained of; the procedure has the additional advantage of producing the desired effect in less time than an emetic; the evacuation of the stomach-contents, moreover, is more complete and more certain.

Many patients are afraid of the tube, and protest if the very name of the instrument is mentioned. If the instrument is unskilfully introduced, the patients undoubtedly suffer a great deal of distress, and it is the duty of every physician to practise the introduction of the stomach-tube until he becomes an expert in its manipulation. Very few patients will object to the introduction of the tube if the physician insists upon it energetically.

Its use is still less common in children. In the case of very small children, where acute gastritis is very frequent, it is still customary to give emetics. This method is certainly insufficient; the administration of an emetic never produces complete evacuation of the stomach-contents, whereas the use of the tube does. In the case of children it is particularly necessary to do something at once, for severe symptoms of collapse may appear suddenly in acute gastritis. It is astonishing to see the good effects that almost immediately follow thorough lavage of the stomach. Although the children may have vomited repeatedly, there is no improvement in their condition, as only a small portion of the ingesta is removed with each effort, and the stomach is never completely evacuated. As a result, fermentation and abnormal decomposition of stomach-contents continue. As soon, however, as the stomach is thoroughly washed out, vomiting stops and the child rapidly recovers. In passing the tube in children, a size should be chosen that is adapted to their age, and in very small children a Nélaton urethral catheter[1] should be used.

In performing lavage of the stomach it is best to instruct the patient to occupy different positions, for only in this way can the organ thoroughly be cleansed. The water should first be allowed to flow when the patient is in the erect position; then when he is lying down. In the case of small children this is not difficult; in the case of adults it is necessary that the patient should have a certain tolerance for the tube; if this is not the case, the instrument, of course, cannot remain in place a sufficient length of time. If necessary, lavage may be repeated. It is also good sometimes to add a little sodium bicarbonate (a teaspoonful to one liter) to the wash-water, for this aids in dissolving the mucus. Some authors have recommended the addition of hydrochloric acid in order to destroy fermentative organisms. From a theoretic point of view this procedure is rational; in practice, however, I have never seen any appreciable effect on fermentation following the employment of such small quantities of hydrochloric acid as are used in this treatment. The most important point is thoroughly to wash the stomach, if necessary, in obstinate cases, repeatedly.

Medicamentous treatment proper is hardly indicated in cases of simple acute gastritis. If the case is one of poisoning and there is severe gastritis, drugs are necessary. I refer to the section on the different forms of intoxication for the different antidotes that are required.

Diet.—As in any other form of acute inflammation, the affected organ should be spared and kept at rest. This can very easily be done in the case of the stomach—more easily, in fact, in the case of this organ than in any other. In small children, particularly if they are very much exhausted after an attack of acute gastritis, it may be good treatment to give a little food soon afterward. In the case of adults, however, it is an easy matter to maintain total abstinence for one or two days. If necessary, food can be introduced in some other way; this, however, is rarely necessary. The chief reason why this total abstinence treatment is not more generally carried out in practice is that the patients themselves believe that the stomach must be offered food, and that they will be cured sooner if they eat something. As a matter of fact, nature seems to point the way, for, in general, there is so much nausea that the patients cannot eat anything. Physicians should combat this prejudice and see that nothing is taken. It is utterly senseless to prescribe a remedy *ut aliquid fecisse videamur;* this is particularly the case if the administration of medicine interferes with the rest that the stomach should enjoy. Occasionally the exhibition of some drug may be necessary in order to act psychically or by suggestion. If the patients are prejudiced in favor of eating something, they should be advised to refrain from doing so for a day or two; they must be convinced that the introduction of food is bad. It is much better to instruct the patients to do absolutely nothing, and not to eat anything at all, than to try different things in order to determine what the stomach will tolerate. The length of time during which the patient should abstain will vary with the severity of the attack. In simple acute gastritis one or two days of abstinence are usually sufficient. The patients themselves will usually follow out the treatment much more readily than the relatives, chiefly because the former suffer from anorexia and do not care to eat.

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[1] It is true that coarse, coagulated particles of food cannot be removed completely through a Nélaton catheter; the introduction of the instrument, however, causes vomiting, and it is an easy matter thoroughly to cleanse the stomach afterward by washing it with water.

Whereas the patients rarely have a craving for food, they frequently complain of thirst. It is not good treatment to allow them to drink large quantities of fluid; the patients may be allowed to moisten their mouth, to rinse it with water, or to hold little pieces of ice in their mouth without swallowing them. Small quantities of carbonated water or a dilute solution of hydrochloric acid may be allowed. Sometimes the patients enjoy a little cold unsweetened tea. The patients should always be instructed to take very small quantities of these different beverages at one time.

If this treatment is instituted, acute gastritic symptoms usually disappear in a short time; the patients regain their appetite, and the tongue, which is usually thickly coated, soon clears up. In the beginning liquid food should be given, particularly soups and mushes, but only in small, gradually increasing quantities. A little yolk of egg may be added to these soups. Milk taken a swallow at a time is also tolerated. As a rule, the appetite rapidly improves, so that a larger quantity of solid food can be administered; I have formulated a scale of digestibility in the section on Diet. The patients may have some young veal boiled in bouillon, calves' brain, boiled pigeon, boiled chicken, scraped raw beef, scraped smoked ham, etc.; in addition, some cakes, a little Zwieback, and later a little mashed potato.

In many cases, particularly the mild ones, the treatment described is all that is necessary. In some cases, however, other methods will have to be instituted. Many cases of gastritis develop symptoms of intestinal inflammation; this is evident when we consider that the same noxious influences that irritate the stomach usually irritate the intestine as well; when both stomach and intestine are irritated, we usually speak of acute gastro-enteritis. If we have reason to believe that the intestine also is inflamed and that decomposed and irritating masses are present, it is good treatment to evacuate the bowel-contents. Simple injections of water are not sufficient, because they reach only the lower portion of the intestine. Calomel is the best remedy, and is successful in treating the gastro-intestinal disorders of children; it should also be employed in the case of adults. This drug is particularly suitable because it leads to an easy and rapid evacuation of the bowel-contents, possesses cholagogue properties, and is at the same time a disinfectant. Simple constipation can usually be treated by large injections of warm water. Only in those cases where obstinate constipation persists for several days, and where repeated injections of warm water fail to give relief, laxatives may be employed; violent purgatives and drastics should be avoided. The best remedy in these cases is castor oil.

Violent attacks of diarrhea are rare. It is bad treatment to try to stop them at once by the administration of opiates; it is much more rational to promote a thorough evacuation of the intestine by the administration of a few large doses of calomel, particularly in those cases in which diarrhea occurs in the beginning of the attack.

Of other symptoms that occasionally need special treatment I might mention the loss of appetite. If anorexia persists for a long time, hydrochloric acid may be administered to advantage. This remedy should be given in doses of from 8 to 10 drops in a wineglassful of water before eating. I have found that the administration of hydrochloric acid after meals is not so useful as before eating, and that the former mode of exhibition does not improve the appetite. The theory of administering hydrochloric acid before meals is, of course, to supply free hydrochloric acid, which is usually absent in acute gastritis. Condurango bark in the form of the decoction of the maceration, plus a little hydrochloric acid, or in the form of the fluid extract, may be tried in these cases.

Analgesics are rarely indicated. Pain, which occasionally occurs together with the symptoms of gastritis, with nausea and vomiting, is usually relieved by the other measures that we have described, particularly by thorough lavage of the stomach. If pain persists for a long time after the stomach has been washed out, the diagnosis of acute gastritis becomes doubtful. In some cases there is an extraordinary degree of hyperesthesia, and here it may be necessary to administer sedative or narcotic remedies. It is better not to give these drugs by mouth, but to administer them in the form of suppositories. The best drugs for this purpose are belladonna, codein, or opium. The subcutaneous injection of morphin cannot be recommended.

(*b*) SEVERE ACUTE AND TOXIC GASTRITIS.

The majority of authors prefer the term "toxic gastritis," and this designation is justified chiefly for the reason that severe forms of acute gastritis are probably always due to the action of some toxic substance. My reason for using the term "severe gastritis" is twofold: in the first place, I wish to place this form in juxtaposition to simple acute gastritis; in the second place, I wish to indicate that every form of toxic gastritis is not necessarily severe. Penzoldt, as a matter of fact, goes so far as to say that almost any form of gastritis is more or less toxic in character, and I agree with him. It is almost impossible to differentiate the different forms of gastritis according to their etiology. For all these reasons the classification of acute gastritis into mild and severe forms seems to be the most natural one, even though the term toxic gastritis is justifiable from a practical point of view.

It will, of course, be impossible to discuss all the symptoms of intoxication that are produced by different poisons; all we can do in this place is to discuss briefly the more severe gastric symptoms that are produced by certain toxic substances. The poisons that are capable of directly damaging the gastric mucosa are, above all, concentrated mineral acids, carbolic acid, caustic alkalis, and alcohol. Phosphorus, arsenic, corrosive sublimate, potassium chlorate, cyanid of potassium, and similar poisons may also produce the symptom-complex of severe acute gastritis.

Mineral acids and caustic alkalis are the most prolific cause of these severe forms of gastritis. The symptoms will vary according to the character of the poison, according to the quantity ingested, and according to the state of the stomach when the poison was introduced. If the stomach was empty, the effects will be more severe than if the stomach was filled with food.

Some toxic substances produce varying degrees of sloughing of the gastric mucosa; others produce inflammatory irritation or glandular inflammation, with fatty degeneration of the glandular epithelia, etc. The former effect is exercised chiefly by corrosive poisons, namely, acids and caustic alkalis; the latter, by alcohol and phosphorus.

Symptoms.—As soon as any of the above-named poisons come in contact with the gastric mucosa symptoms of violent acute gastritis appear, followed sooner or later by serious symptoms of general intoxication. In regard to the latter group of symptoms, we refer to the section on Intoxications. If corroding fluids are swallowed, much will depend on the position that the patient occupies at the time; whether or not the pharynx and the esophagus are affected. If the corroding fluid is taken while the individual is in the erect position,—and this will be the rule,—the region of the lower end of the greater curvature near the pylorus will chiefly be involved. This explains the fact that swallowing corrosive poisons frequently leads to cicatricial stenosis of the pyloric region; if the poison is swallowed while the patient is lying down, the pharynx and esophagus are injured to a greater degree than if the patient is sitting down or standing up. At the same time the first effect of the poison will be exercised on the posterior wall of the stomach.

Immediately after swallowing the poison, whatever its character, the patient experiences pain in the pharynx, along the sternum, and in the epigastric region. The pain is usually very violent and burning in character. Almost without exception vomiting occurs and is frequently repeated. Even this, however, fails to relieve the patient's distress. If the poison enters the stomach when it is empty, vomiting usually occurs at once; if the stomach was full, vomiting usually is delayed a little while. The appearance of the vomit varies accordingly.

If the stomach contained food, abundant food-remnants will be found in the vomit; if not, there will be much blood and mucus, and possibly shreds of the mucous lining of the stomach.

External inspection of the abdomen reveals nothing characteristic. In some cases the region of the stomach protrudes; in others it is retracted. In all cases the abdomen, and particularly the gastric region, is sensitive to pressure—sometimes exceedingly sensitive and painful to the slightest contact. The facial expression is anxious and suffering, and the color pale; the forehead is covered with cold perspiration; the pulse is weak, easily compressible, and greatly accelerated. The extremities, particularly the hands and feet, are cool, cyanotic, and covered with cold perspiration. Respiration is accelerated, superficial in type, and chiefly thoracic; this is due principally to the fact that the patients try to limit the excursions of the diaphragm, for every movement of the midriff causes violent pain. In very severe cases there may be perforation and peritonitis; in other cases again the patients soon fall into a state of deep collapse, even though no perforation occurs. This fulminating acute course is seen only in very severe forms of intoxication, particularly after swallowing mineral acids or caustic alkalis.

Many degrees of toxic gastritis are seen, varying in severity from the violent and the acute form that we have described, which lead to the death of the patient within a few hours or a few days, to the very mild forms of gastritis that we have described in the preceding paragraphs. In many cases, for instance, very violent gastric symptoms with vomiting and gagging appear immediately after swallowing the poison, but all these symptoms disappear in the course of a few hours and the patient is restored completely within a few days. In other cases again the violent gastric symptoms disappear after a short time, but symptoms of blood-poisoning or of general intoxication appear in their place; even if the patients recover from the latter, certain complications persist that may lead to permanent disorders, or, after some time, to the death of the patient. I do not refer alone to stricture of the esophagus, which does not properly belong under this heading, but chiefly to atrophy of the mucous lining of the stomach and cicatricial stenosis of the pyloric region. This atrophy of the mucosa may be exceedingly severe, so that the production of hydrochloric acid is damaged to a great extent. I[1] demonstrated this in a case that I reported not long ago. The patient furnished so typical an illustration of the results of a severe attack of gastritis that I think it will be useful to describe the case briefly in this place.

The patient was a day laborer of forty-eight years, who was received in my clinic on the second day of July, 1885. He stated that six weeks ago he had taken a drink from a whisky bottle that had been filled with sulphuric acid by some malicious person. He claimed not to have felt any particular burning in the mouth, but violent pains and a feeling of pressure in the stomach. Since then he vomited everything he ate.

The patient was greatly emaciated and cachectic. No scars were found on the tongue nor on the tissues of the pharynx. The stomach was much distended, and showed peristaltic movements from time to time, the waves extending from left to right. No tumor could be felt. The stomach was pumped out daily and vomiting stopped, but the patient continued to lose flesh. He died within a month after his admission. The stomach was washed out 31 times in all, and abundant quantities of food were always found late in the evening, particularly coarse and undigested particles of meat, but no free hydrochloric acid. Organic acids were always present. The peptic powers of the filtrate of the stomach-contents were so small that a flake of albumin was never digested.

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[1] Riegel, *Zeitschr. f. klin. Med.*, vol. xi., Nos. 2, 3.

Professor Bostroem performed the autopsy and reported the following: The stomach was dilated; the pyloric ring contracted. Near the greater curvature and opposite the cardiac orifice of the stomach there was a sharply defined and clearly circumscribed area of ulceration. At this place the surface of the stomach presented a very peculiar appearance. There seemed to be two kinds of tissue in close proximity to each other: first, broad, grayish-white bands; second, a large number of minute, almost punctiform deposits arranged in striæ and branching out in different directions; the latter were of a grayish-yellow color, soft, and could readily be rinsed off with water.

Microscopic examination of the ulcerated spots showed that the mucous membrane was lost, with the exception of a few small shreds that were still present in isolated places; these shreds of membrane corresponded to the brownish-yellow deposits that we have described above. The rest of the surface of the ulcer was formed by thick layers of coarse connective tissue that extended downward to the muscularis; the latter was also somewhat thickened.

In this case, therefore, there had been a transitory toxic gastritis that was in process of repair; there had been formation of scar-tissue that had led to stenosis of the pylorus and secondary ectasy. The absence of free hydrochloric acid and the great reduction in the peptic powers of the gastric juice were readily explainable from this defect in the continuity of the mucosa. The fatal issue was undoubtedly precipitated by the lesion and the stenosis of the pylorus that was caused by it, the latter condition, of course, preventing the propulsion of the ingesta into the intestine within the normal time.

This example is a typical illustration of the course of a gastritis that is produced by mineral acids. The general disease-picture will, of course, vary in each individual case according to the poison. For a description of all these variations I must refer the reader to the sections on the different forms of poisoning.

Anatomic Findings.—The anatomic lesions observed may also vary greatly. Phosphorus, alcohol, and arsenic produce severe degrees of hyperemia and swelling of the mucosa; occasionally even large and small hemorrhages. None of these features, however, is characteristic for the poisons enumerated. The most typical process that follows intoxication with phosphorus, alcohol, or arsenic is a pronounced fatty degeneration of the glandular epithelium, which has a tendency to spread over large areas of the mucosa. This condition has been called gastritis glandularis (Virchow). The most typical and the most pronounced picture of fatty degeneration of the glandular parenchyma of this character—of so-called parenchymatous degeneration—is seen in phosphorus-poisoning.

The action of corroding poisons, as mineral acids and caustic alkalis, on the gastric mucosa is different, as we have already seen. In this form of intoxication, particularly if the poison is very concentrated, the lesions observed are much more extended; in the beginning the mucous membrane will be found swollen and hyperemic, and covered with numerous hemorrhagic spots; later there will be sloughing that may pass through the whole mucous lining or even extend to the deeper tissues; occasionally the wall of the stomach may even become perforated. If the effect of the poison is not too violent,—*i. e.*, if the patient survives,—the diseased portion of the stomach sloughs off; this may lead to hemorrhages, but, as a rule, cicatricial connective tissue develops; this, later on, contracts and leads to cicatricial stenosis of the stomach, or produces various changes in the form of the organ—as, for instance, hour-glass contraction, etc.

If large portions of the gastric mucosa are destroyed, as in the case described above, there will be serious perversions of gastric secretion and ectasy. If the poison enters the stomach in a very dilute form, the changes observed will, of course, not be so severe. As a rule, the clinical picture of a simple gastritis of short duration develops in these cases.

Diagnosis.—The diagnosis of severe or toxic gastritis is, as a rule, easy. The sudden appearance of very violent gastric symptoms in a perfectly healthy subject should always lead us to suspect that some form of poisoning has occurred.

In many cases the diagnosis is rendered easy by the history of the case, the patient stating that he has swallowed some poisonous substance. This is always the case where poisoning occurs from some unfortunate mistake; in other cases, again, the nature of the poison that has been swallowed can be determined by an analysis of the stomach-contents or of the contents of the receptacle from which the substance was taken; in other cases, again, a careful examination of the lips, the mouth, and the pharynx may furnish valuable clues; we may learn that an irritating substance or an eroding substance has passed through the organs of deglutition—in fact, the appearance of these parts frequently enables us to gain some idea of the nature of the poison. Again we must refer to the sections on poisoning for the details.

Treatment.—The first object in treating a case that has swallowed some toxic substance is to remove as much of the latter as possible, or to neutralize it. Although in the majority of cases of poisoning violent vomiting follows the ingestion of the poison, a certain proportion of the substance usually remains behind. Emetics, if they can be given at all, rarely produce a complete evacuation of the stomach-contents. The passage of the stomach-tube is much more rational, for by this means we succeed in removing all the toxic substance that remains in the stomach, and can, at the same time, introduce the necessary antidotes into the stomach within the shortest time and in the most certain way. This method, of course, is applicable only in a certain proportion of cases, and may even be contraindicated. The sound may be passed whenever the poison that has been swallowed is not one of a corroding nature. Its passage is contraindicated, or is at least dangerous, where mineral acids or caustic alkalis have been swallowed, for here there is always danger of injuring the gastric mucosa, particularly in those spots where it has become eroded.

If it is impossible to introduce the sound, or if there is some contraindication to its passage,—in other words, if the remainder of the toxic substance cannot be removed from the stomach by aspiration,—antidotes will have to be administered. I refer to the section on Intoxication for the antidotes that are indicated in different poisons. If the sound can be passed, the antidote can be administered through it.

The treatment of severe acute gastritis is based on essentially the same principles as the treatment of simple acute gastritis. In the former class of cases treatment must be instituted for a longer time, and the stomach must be kept at rest and spared still more conscientiously. In many cases of this character it is necessary to feed the patients by rectum for a long time. In other instances certain sequelæ have to be considered and treated. These consist essentially in atrophy of the gastric glands following wide-spread destruction of the gastric mucosa; here the peptic powers of the stomach will be found greatly reduced. Then there are cases of cicatricial stenosis, particularly of the pylorus, that have to be treated. As long as the patient is merely afflicted with a deficiency of hydrochloric acid secretion, and as long as the motor power of the stomach remains normal, a compensation of the deficiency existing can usually be brought about to a certain degree. In these cases the deficiency in peptic power is usually compensated by the more rapid propulsion of the ingesta into the intestine. As a rule, however, the tone of the stomach is more or less reduced and there is a cicatricial stenosis of the pylorus, so that the propulsion of the gastric contents into the intestine is hindered. As the material remaining behind is only partially digested or not digested at all, putrefactive and fermentative processes develop, so that the material, when it ultimately does enter the intestine, is in such a state of decomposition that intestinal digestion cannot take place, and the compensatory effect of the latter process is lost.

In all these cases the only remedy is an operation. Surgical procedures cannot, of course, reëstablish the peptic powers of the stomach, but they can aid the more rapid propulsion of the ingesta into the intestine. The best operation for this purpose is gastro-enterostomy; in some instances pylorectomy or pyloroplasty may be considered. There are two objections to the former operation, namely, that it is very much more difficult to perform than the latter, and that it frequently proves unavailing. In many cases, besides, there will be a recurrence of the primary trouble.

Another surgical procedure must always be considered in dealing with cases of severe toxic gastritis in which perforation has occurred, namely, immediate laparotomy. The only way to save the patient in these cases is to open the abdomen and close the peritoneal wound.

(*c*) GASTRITIS PHLEGMONOSA; INTERSTITIAL SUPPURATIVE INFLAMMATION OF THE STOMACH.

LITERATURE.

For the older literature I refer particularly to Leube's *Diseases of the Stomach* in von Ziemssen's *Hand-book of Special Pathology and Therapy*; also, to the summaries of Raynaud, etc.

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Dörbeck, "Ein Fall diffuser eiteriger Entzündung des Magens," ibid.
Kelynack, "A Case of Diffuse Phlegmonous Gastritis," Lancet, 1896.

Compare also the well-known text-books on diseases of the stomach by Boas, Bouveret, etc.

Introductory Remarks.—Gastritis phlegmonosa is one of the rare diseases of the stomach. The condition is characterized by a purulent inflammation of the stomach-walls that usually starts from the submucous tissues and leads to more or less extended suppurative degeneration of this portion of the stomach-wall. Occasionally it involves the mucosa, the muscularis, the subserosa, and the serosa. It may occur as a primary disease of unknown origin, or it may be secondary. In the latter case it is usually due to a metastatic process following puerperal infection and pyemia. In both the primary and the metastatic form we must assume that pathogenic micro-organisms enter the submucosa and directly cause disintegration of this portion of the stomach.

Phlegmonous inflammation of the stomach may further be subdivided according to its extent. We distinguish two forms: first, diffuse phlegmonous gastritis, in which the purulent infiltration extends uniformly over a large portion of the stomach; and, second, the circumscribed form, so-called abscess of the stomach. Of the two forms, the former is relatively more frequent; but even diffuse phlegmonous gastritis is a rare disease, for there are only some 50 cases on record. In 1892 Mintz[1] published a summary of all the cases reported up to that time. This summary included 43 cases, and very few have been reported since then.

Anatomic Changes.—In diffuse suppurative inflammations of the stomach anatomic changes are seen either throughout the greater portion of the stomach or in a small circumscribed area. As a rule, the region of the pylorus is the one most seriously involved. If a transverse section is made through the stomach-wall, it will be found that the whole wall, and particularly the submucosa, is thickened. A purulent or seropurulent fluid will be seen to ooze from the cut surface. Pus is absent only in those cases in which the stomach is examined in the very beginning of the disease. One case is on record in which the purulent infiltration of the stomach was revealed only by microscopic examination. This case was reported by Hilton Fagge.[2]

The principal changes are seen in the submucous tissue. The submucosa is broader than normal, shows purulent infiltration throughout its whole extent, and is saturated with a seropurulent or purulent fluid. In more advanced cases the submucosa may be completely destroyed and the space between the mucosa and the muscularis occupied by a layer of pus. The purulent inflammation may include the muscularis, so that the walls of the pus cavity are formed, on the one side, by the serosa, on the other, by the mucosa. In the majority of cases the mucous lining itself is involved, though usually only to a slight degree. The inflammation extends between the glandular tubules and involves the interglandular tissues. The mucous lining is, as a rule, thickened, hyperemic, covered with ecchymoses, and the glandular epithelia are granular or in a state of fatty degeneration; the intraglandular tissue shows cellular infiltration. In some of the cases the pus has been known even to perforate certain portions of the mucosa, so that the latter membrane appeared like a sieve. Large and small ulcers are also occasionally seen. In two cases (Gläser[3] and Mintz[4]) carcinoma of the stomach was present at the same time.

The muscularis, in the majority of cases, remains intact. Here and there areas of small-celled infiltration are seen between the different strands of muscle tissue and the latter are forced asunder, so that the muscle fibers in part undergo fatty degeneration. In a few instances the pus burrowed as far as the serosa and perforated the serous lining of the stomach. A few cases are on record in which the serosa became adherent to neighboring organs.

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[1] Mintz, "Ein Fall von Gastritis phlegmonosa diffusa im Verlaufe eines Magenkrebses," *Deutsch. Arch. f. klin. Med.*, vol. lxix.

[2] *Virchow-Hirsch's Jahresb.*, 1876, vol. ii., p. 225.

[3] Gläser, *Berlin. klin. Wochenschr.*, 1883, No. 51.

[4] *Loc. cit.*

Further consequences of purulent peritonitis are extension of the purulent inflammation of the stomach-wall to the first portions of the duodenum or the lower portion of the esophagus.

The streptococcus has been found in the submucosa in a number of cases, and was probably the cause of the disease. [A classical case of idiopathic phlegmonous gastritis was reported by Kinnicutt[1] in which the submucosa was enormously thickened, measuring in certain places $\frac{3}{4}$ of an inch, even in the hardened sections. The muscle-fibers were swollen and distorted, the subserous layers were irregular, thickened, and infiltrated with round cells. The infection was found to depend upon the streptococcus. The patient died on the sixth day of illness, during the first three of which he suffered pain, made worse by vomiting; but for two days preceding his death he was comparatively free from suffering. The upper part of the abdomen was moderately distended and resisting. Respiration 48, pulse 108, rectal temperature 101° F.—Ed.]

Abscess of the stomach proper presents a different appearance. Usually a pus cavity is found in the submucosa that may vary in size and may be as large as an adult's fist. As a rule, the muscularis and the subserosa are also involved. The serosa and mucosa may be intact; as a rule, however, they are very much inflamed. Abscesses of this kind may be either single or multiple; they may perforate into the cavity of the stomach or through the serosa into the peritoneal cavity. Occasionally a restitution to normal occurs, with the formation of scar-tissue. Such a favorable issue occurs only in small pus foci.

Etiology.—The etiology of this disease is obscure. It has been found more frequently in men than in women. The following etiologic factors have to be considered—alcoholic excesses, errors in diet, "catching cold," traumata. The disease is not caused directly by these different agencies; it is more probable that there is a dissolution of continuity somewhere in the lining membrane of the stomach through which pus-organisms penetrate the submucosa.

The etiology of phlegmonous inflammation of the stomach is less obscure in the secondary metastatic forms that follow puerperal infection and pyemia, and occasionally certain other infectious diseases.

Symptoms.—The disease-picture of phlegmonous gastritis is not at all typical. Mintz distinguishes two periods of the diffuse form of the disease: a first period, that terminates when peritonitis occurs; and a second period, in which the peritonitic symptoms are the most important. In general the symptom-complex corresponds to that of a severe gastritis, with high fever and severe general symptoms. Vomiting occurs in the great majority of cases; as a rule, it occurs in the beginning of the disease and continues throughout its course. In those cases of phlegmonous gastritis that are complicated with carcinoma of the stomach vomiting seems to be absent. This is a very striking observation, particularly as vomiting is usually present in carcinoma before gastritis develops.

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[1] *Trans. Assoc. Amer. Phys.*, 1901.

Vomiting usually leads to the evacuation of food-remnants, mucus, and bile; pus has never been found in the vomit. There are usually nausea, anorexia, and increased thirst. Most patients complain bitterly of violent pain in the epigastric region that usually extends over the whole area occupied by the stomach, and even beyond the boundaries of the organ. This pain is usually increased by pressure, although some observers expressly emphasize the fact that this symptom was absent. The abdomen is usually meteoristic and the bowels constipated. In a few cases there were severe diarrheas. The temperature-curve and the general symptoms are also important; fever occurs from the very beginning, and may rise as high as 40° C. and higher, and persist throughout the disease. There is nothing typical or characteristic about the temperature-curve. The pulse is small, very much accelerated, and in the later stages occasionally irregular. The general health of the patient is seriously impaired soon after the onset of the first symptoms. As a rule, the patients are apathetic, they lie quietly in their bed, and soon fall into a condition of stupor that is interrupted by delirium; occasionally they seem to be more excited, are restless, and complain of precordial distress. As a rule, they soon die in collapse. In other cases perforation occurs and the symptoms of peritonitis appear.

The disease-picture of abscess of the stomach does not differ from the picture of phlegmonous gastritis that we have delineated, in any essential point; the only difference between the circumscribed and the diffuse form of phlegmonous gastritis is the more protracted course of the former disease. Here, too, certain gastric symptoms usher in the disease; these may be very mild in the beginning, but soon increase in severity. Violent pain and frequent vomiting soon supervene; the pain is usually circumscribed and limited to the region of the stomach; it rarely radiates. Pressure over the stomach increases it. A tumor can only rarely be felt in the region of the stomach; all the other symptoms are essentially the same as those described in the diffuse form: we have fever, acceleration of the pulse, and severe general symptoms. Most of the patients die in a short time in a state of collapse. The course of the disease is rarely protracted; occasionally the patients may linger for several weeks. If peritonitis occurs from perforation, meteorism, collapse, and death occur in very short order. It has not been positively determined whether or not this condition is occasionally cured. Dittrich reports the presence of cicatricial tissue in the submucosa in a few cases that he examined; this seems to indicate that small abscesses of the stomach-wall may occasionally be cured.

Diagnosis.—A diagnosis of diffuse and circumscribed phlegmonous gastritis can rarely be made with certainty; the symptoms, it is true, point to some severe infectious disease with violent gastric symptoms, but no characteristic signs appear. As a rule, the diagnosis of peritonitis is made, as this condition is frequently present; it is rarely possible, however, to state with certainty that peritonitis originates from a phlegmonous gastritis; the severity of the symptom-complex and the rapidity with which the different symptoms appear may lead to the suspicion of toxic gastritis; as a rule, however, the history of the case will furnish no information that would justify this diagnosis, no evidence of corrosion will be found in the mouth, examination of the vomit will yield negative results, so that this diagnosis can generally be excluded.

Pus frequently perforates into the stomach, and one should think that it would frequently appear in the vomit; so far, however, pus has been found in the vomit in only one case,[1] and this case was not examined postmortem.

But even if pus were more frequently found, the diagnosis of phlegmonous gastritis could not be positively made. Leube[1] has reported a case of acute purulent gastritis in which this was very distinctly seen. In this patient all the principal symptoms of phlegmonous gastritis had developed. The disease-picture was a very severe one: the patient was in collapse, there were violent pain in the stomach region, intense vomiting, fever, and a small, irregular, accelerated pulse. The vomit contained numerous stomach epithelia, an enormous quantity of mycelia and bacteria, and some pus. Nevertheless no submucous gastritis was found postmortem, but only a very severe inflammation of the gastric mucosa. There was an exceptional amount of purulent secretion on the free surface of the stomach-wall. The diagnosis might possibly be made if the symptom-complex that we have described appeared in the course of some severe infectious disease, as puerperal fever, pyemia, etc.

In the case of stomach abscess the same diagnostic difficulties obtain as in diffuse phlegmonous gastritis. In both forms of purulent inflammation of the stomach-wall the same symptoms appear. The diagnosis might possibly be made if a tumor were felt in the stomach-wall that became reduced in size or disappeared after the vomiting of pus, but even in a case of this kind the condition might be confounded with an abscess of some neighboring organ that had perforated into the stomach. We may say, therefore, that the diagnosis can probably never be made with certainty.

Prognosis.—The prognosis, it may be said, is almost altogether unfavorable. Dittrich, as I have already said, found cicatricial tissue in the submucosa in two subjects, and claimed that this demonstrates that purulent infiltration of the parts existed at one time, but was cured. So far these cases are isolated, and the interpretation that Dittrich gives is by no means free from objection.

Treatment.—The treatment must be more or less symptomatic, if for no other reason than that the diagnosis is never positive; but even if the diagnosis should be made with certainty there would be no successful treatment of this disease.

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[1] This patient that Kirschmann (*Wien. med. Wochenschr.*, 1880) reported was a drunkard of twenty-five years. He fell on his stomach, and immediately complained of very violent pain and vomiting. At the end of seven days half a liter of pure pus was vomited. All the symptoms immediately disappeared after this and the patient recovered.

The same methods will have to be employed in the treatment of the severe gastric disturbances that are useful in the other forms of gastritis that we have previously described. The chief indication will be to spare the stomach. The introduction of food and drink should proceed by rectum. For the pain, narcotics may be given. Antiphlogistic measures, like cold compresses, ice compresses, ice-bags, etc., should be employed, particularly as the symptoms are similar to peritonitis or may even be due to peritonitis. Stimulants must, of course, be administered early in the disease, and are called for as soon as symptoms of collapse appear.

(*d*) CHRONIC GASTRITIS; CHRONIC CATARRH OF THE STOMACH.

Introductory Remarks.—Formerly chronic catarrh of the stomach was considered the most frequent disease of the stomach, and even to-day this belief is prevalent; as a matter of fact, however, chronic catarrh of the stomach is not a frequent disease.

In 1878 Leube, in his well-known hand-book on *Diseases of the Stomach*, formulated the following rule: Never to diagnose primary catarrh of the stomach unless all other possible chronic diseases that can produce dyspeptic symptoms can be excluded with certainty. This dictum is valid to-day, with this reserve, however, that the exclusion of all other diseases is based on modern methods of examination and not, as formerly, on a congeries of subjective symptoms and the older methods of examination.

Modern methods of examination have taught us to recognize a number of new typical disease-pictures; as a result, we have learned to recognize a number of characteristic forms of stomach disease that we classify as typical functional perversions. However valuable the methods of diagnosis by exclusion may be, we should not content ourselves with purely negative evidence. This applies particularly to diseases and functional perversions of the stomach, for in the case of this organ clinical methods of examination are still in a rudimentary stage.

Many symptoms that we are forced to-day to interpret as simple perversions of function may some day be found to be due to well-characterized anatomic changes in the stomach. A careful anatomic examination of the stomach is so difficult that we need not be surprised to find our knowledge in this respect very deficient; it seems more rational, therefore, to give up the attempt to forcing all known disorders of the stomach into a few disease categories; it is much better simply to record them as objective clinical findings. If we agree to proceed in this way, we can speak of chronic gastritis only when we find positive signs of this condition; and we shall soon learn that the disease is not so frequent as older physicians thought, and as many modern ones still persist in believing.

Many authors subdivide chronic gastritis. The different subdivisions really constitute differences in degree, not in type. From a practical point of view I think we are justified in distinguishing two forms: (*a*) chronic gastritis, and (*b*) atrophy of the mucous lining of the stomach.

[The eminently correct views above expressed as to the comparative infrequency of chronic gastritis will be indorsed by experience if the cases are carefully studied. The error is so commonly made of ascribing to chronic gastritis the symptoms that depend upon a variety of other affections, that we may be excused for emphasizing the clear and strong statements bearing on this part of the subject.—Ed.]

Etiology.—All agencies that are capable of causing acute gastritis may lead to chronic gastritis provided they act for a sufficiently long time or are repeated a number of times.

We distinguish a primary and a secondary form. In the former case the damage affects the stomach directly; in the latter, gastritis develops as a result of some other disease. Chronic gastritis, particularly the primary form, is encountered more frequently in men than in women; this is due to the fact that the former are more exposed to the agencies that may cause gastritis than the latter.

Primary chronic gastritis may develop from acute gastritis; as a rule, this mode of development is rare. General acute gastritis is an isolated event due to the action of some one noxious agency. If appropriate treatment is instituted in good time, acute gastritis is usually completely cured. In order that chronic gastritis may develop, some disease of the stomach must exist for a relatively long time; we may say, therefore, that chronic gastritis begins gradually. In many instances a predisposition to diseases of the stomach, particularly gastritis, exists from childhood up, and is due, as a rule, to irrational mode of life. If the diet is not well selected; if the patients eat too rapidly and do not masticate their food with sufficient thoroughness, gastritis may develop. The disease is often seen in old people who have lost a large number of teeth and cannot chew their food. The best prophylactic in these cases is a well-fitting set of teeth. The majority of people underestimate the significance of thorough mastication and thorough insalivation of the food. Sticker has performed a large number of investigations on the rôle of mouth digestion on digestion in the stomach, and has shown that the former exercises a marked effect on the latter.

Frequent overloading of the stomach with indigestible food, frequent excesses in eating and drinking, will lead to chronic gastritis in the same way as deficient mastication of the food.

Many authors speak of a hereditary predisposition to gastritis. It is true that a certain weakness of the stomach may be inherited; but the chief part in the causation of gastritis is played by coddling the stomach and living in an irrational manner.

One of the most important etiologic factors in the production of chronic gastritis is the abuse of alcohol. Drunkards' catarrh is one of the most frequent forms of gastritis that is observed. The form in which alcohol is taken is quite important. Chronic gastritis is seen more frequently in whisky drunkards than in those who overindulge in wine and beer. The excessive use of tobacco, particularly chewing-tobacco, frequently leads to gastritis, although every attack of dyspepsia that occurs in smokers is not necessarily due to gastritis. Coffee and tea have also been accused of predisposing to the development of gastritis, and in the same way strong spices, certain drugs, and the excessive use of purgatives.

In the second group of cases gastritis is secondary, and may be the result either of some general disease or disease of some one organ.

Certain diseases of the stomach itself belong to the latter category, for many of the lesions of the stomach may secondarily lead to chronic gastritis. Carcinoma of the stomach, for instance, frequently causes catarrh of the mucous lining of the organ. Ulcer, as far as I have seen, rarely leads to catarrh of the stomach, although many authors are inclined to regard ulcer of the stomach as one of the diseases that usually lead to secondary gastritis. Ectasy and atony of the stomach frequently cause chronic gastritis, for here all the conditions for the development of such a state are given. Gastritis, however, does not necessarily follow every case of ectasy and atony. Occasionally diseases of remote organs lead to chronic catarrh of the stomach, particularly by interfering with the circulation of the organ; thus we see chronic gastric catarrh in certain diseases of the heart and of the liver, in affections of the portal vein, and less frequently in certain diseases of the kidneys and the lungs.

Among the general diseases, and particularly the diseases of metabolism, serious forms of anemia, chlorosis, leukemia, and diabetes mellitus, are most liable to cause chronic gastritis; but we must not forget that, on the one hand, dyspeptic symptoms are frequently absent in many of these diseases; and that, on the other, every attack of dyspepsia that may occur in any one of these diseases is not necessarily due to gastritis. We will explain in another section how the above-mentioned diseases influence the functions of the stomach.

Pathologic Anatomy.—We have seen that in simple acute gastritis we are hardly ever justified in speaking of a catarrh *per se*. This is still more the case in the chronic form of gastritis. We rarely see changes of the epithelium of the mucosa alone; generally the inflammatory process extends to the glandular parenchyma itself, and occasionally to the interstitial tissues. In some instances the former, in others the latter, are involved; in some cases we have hyperplasia, in others atrophy of the mucous lining. These different forms may gradually merge into one another.

In the ordinary form of simple chronic gastritis the mucous membrane is swollen, thickened, loosened, and of a more or less gray, in other cases dark-red, color. The swelling is not, as a rule, uniformly distributed over the whole mucous membrane, but is generally greater in the pyloric region. The surface of the mucosa is covered with a thick, tough layer of mucus that can be separated only with difficulty; this, too, is most pronounced in the pyloric region.

The mucus is sometimes of a light, translucent, glassy character, but more frequently it is cloudy and gray, owing to the admixture of numerous tissue elements, as cell nuclei, desquamated epithelia, glandular cells, leukocytes, etc. In rare instances the mucus is colored red from the admixture of blood. The mucosa itself, particularly in cases of long duration, looks gray or slate colored; here and there isolated large or small hemorrhagic foci may be seen; in other places again the epithelium may be desquamated so that there are small superficial dissolutions of continuity—erosions, in other words. Microscopic examination presents a still greater variety of pictures than macroscopic examination. The duration of the disease, the character of the process,—that is, whether it is productive of inflammatory or of degenerative changes, whether it involves only portions of the stomach or the whole organ,—change the appearance of the stomach lining in different ways. Within the glands the epithelium is usually cloudy, granular, loosened, in a condition of fatty degeneration, and occasionally contracted and completely atrophied. It is impossible to differentiate the peptic from the parietal cells. The glands themselves are in part broader and larger than normal; they may assume a tortuous course and may undergo cystic degeneration in cases where the exit of the tubules is occluded. In the majority of cases, particularly if the disease is of long duration, the interglandular tissues will be more or less involved in the inflammatory process; here we see an abundant small-celled infiltration and a broadening out of the interglandular tissue; occasionally the latter becomes more vascular and the capillary and lymph-spaces dilate. This infiltration of the interglandular tissues with round-cells causes pressure to be exercised on the tubules of the glands, so that they become distorted and their secretion stagnates. Following this there is occasionally cystoid degeneration of some of the tubules. This proliferation of interglandular connective tissue may finally lead to contractions.

The proliferative processes that we have described are, above all, responsible for the thickening of the gastric mucosa in early stages of chronic gastritis; this inflammatory thickening, however, is not limited to the mucosa alone, but extends to the submucosa if the disease is of long duration and of a certain degree of intensity. The mucosa and submucosa, however, are rarely involved to the same extent; it is due to this that the surface of the mucous lining of the stomach becomes unevenly thickened and folded; sometimes it becomes arranged in transverse and longitudinal folds as a result of this condition. The appearance of the mucous membrane that is changed in this way is very peculiar, and has been called *état mamelonné*. Two factors produce this picture: on the one hand, the proliferation of interstitial tissue; on the other, the hyperplasia of glandular tissue. Under certain circumstances, particularly if the mucous membrane has become more or less atrophic, large wart-like, polypous excrescences may develop that appear either singly or in groups. If they are situated near the pylorus, and if they are very numerous and involve the submucosa, they may lead to obstruction of the pyloric passage; if there is at the same time muscular hypertrophy, the stenosis will be still more serious.

Another form of chronic gastritis has been described that is characterized by sclerotic processes. This condition is rare. The process usually starts from the peritoneum and gradually converts the membranes of the stomach into cirrhotic connective tissue. The epithelium of the mucosa may remain intact. This disease occasionally leads to a reduction in the size of the stomach, with thickening of its walls, so that during life there may seem to be a tumor of the stomach. This form of gastritis has usually been called cirrhosis ventriculi (Brinton) or hypertrophic sclerosis.

We have already stated that in a certain proportion of cases of gastritis the muscular layers of the stomach are not involved; in other instances again there is partial or wide-spread hypertrophy of the musculature. The pyloric region in particular is frequently involved; this may lead to very severe degrees of stenosis, so-called hypertrophic stenosis of the pylorus, in those cases where the mucosa and the submucosa are thickened at the same time. On the other hand, partial degeneration or atrophy of muscle-fibers may occur if the connective tissue between the different muscle-fibers becomes hyperplastic.

The final outcome of gastritis may be atrophy of the mucosa—so-called *phthisis mucosæ* or *anadenia gastrica*. Some investigators claim that a stage of mucoid degeneration of the glandular cells precedes this stage of atrophy, so that immediately preceding the final cessation of the flow of the mucus there is a stage in which the secretion of mucus is very much increased. Schmidt[1] has shown, however, that in this mucous degeneration of gastric epithelium a new form of epithelium is formed that is never normally found in the stomach, and that resembles intestinal epithelium. The cells are cylindric and exhibit a margin. They are closed above, and alternate with goblet cells. They cover the atrophic places on the surface of the stomach and all those places where there is a dissolution of continuity of the mucosa following disintegration of glandular cells.

The peculiar epithelium that Schmidt has described is found at the margin of ulcers of the stomach and in chronic inflammations of the stomach that are accompanied by atrophy of the parenchymatous cells. In the latter instance the peculiar new cells are arranged in little isolated spots. This epithelium, it appears, has the power of secreting much mucus, especially if it contains numerous goblet cells.

In some stomachs large portions of the mucosa will be found covered by this epithelium, even though all the normal cells have perished. For this reason we may occasionally see a large secretion of mucus in a stomach that is in an advanced stage of atrophy. If we find large quantities of tough mucus and no hydrochloric acid or pepsin whatever, we must suspect atrophy (Schmidt[2]). Only in the very last stages of the disease is this secretion of mucus stopped. Occasionally hemorrhagic erosions occur—so-called catarrhal ulcers; these are seen particularly in those cases where the inflammatory processes extend deep down.

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[1] *Deutsch. med. Wochenschr.*, 1895, No. 19.

[2] *Deutsch. Arch. f. klin. Med.*, vol. lvii., p. 78.

The size of the stomach varies considerably in chronic gastritis; in the majority of cases it is of normal size, and only rarely do we find the whole organ contracted and reduced in size. This condition, as we have already seen, is occasionally found in so-called cirrhosis ventriculi; in other cases, particularly if there is hypertrophic stenosis in the region of the pylorus, the stomach may be dilated.

It is almost impossible to recognize the different pathologic-anatomic changes that correspond to the different chronic gastritic processes by clinical methods. Some authors distinguish between chronic and mucous gastritis, but these differences are very artificial and constitute variations of degree and not of kind, for every simple chronic gastritis is a mucous gastritis. The subdivision into simple chronic and atrophic forms is more justifiable, but even these two forms may occur side by side, so that there may be advanced degrees of atrophy in one portion of the stomach and simple inflammation of the mucosa in others. It is, of course, clinically important to determine whether the greater portion of the mucosa is still in a condition of inflammatory irritation or whether it is already in a condition of connective-tissue degeneration and atrophy. For all these reasons I think we are clinically justified in separating simple chronic gastritis from atrophy.

Symptoms.—In rare instances, as we have already seen in discussing the etiology of the disease, chronic gastritis may develop from acute or subacute gastritis. This development is usually slow. The first symptoms are insignificant, but they gradually increase in severity. If patients of this kind are interrogated in regard to the beginning and the course of the disease, they are rarely able to make any precise statements. The general story is that the symptoms were very slight in the beginning, that the appetite gradually decreased, or that it was irregular; they may complain of a feeling of pressure and distress in the region of the stomach; later there may be belching, particularly after eating food that is indigestible. Patients rarely report that there was frequent vomiting in the beginning. All these symptoms gradually increase in severity without, as a rule, becoming particularly intense. None of the symptoms, as we see, is typical or characteristic for chronic gastritis, for we find analogous symptoms in a great variety of other diseases of the stomach.

As soon as the disease reaches a certain height the following symptom-complex is usually presented: the patients complain of a feeling of pressure and fulness after eating, but do not, as a rule, complain of violent pain. There may be belching and nausea, and occasionally vomiting; the latter usually occurs in the morning when the stomach is empty, in some cases, however, at the height of digestion. The patients complain of a bad taste in the mouth, of anorexia, of increased thirst; they feel weak, depressed, and unwilling to perform their duties. They are frequently melancholic and inclined to hypochondriasis. These symptoms are frequently complicated later on by a series of nervous symptoms, so that it is frequently difficult to decide whether the nervous or the gastric symptoms were primary, particularly if the patients are seen for the first time when the disease is fully developed.

All these symptoms may continue at the same height or with varying intensity for many months or years.

We will now briefly discuss the different individual symptoms of chronic gastritis: first the subjective, then the objective.

The appetite is usually more or less reduced in the majority of cases. Frequently the patients feel satiated even after a very small meal. Occasional attacks of violent craving for food seem to occur suddenly, accompanied in many instances by nausea and general malaise; a bite or two of food will usually stop these sensations. Patients of this character frequently have a pronounced desire for acid, strong, or spiced articles of food.

In exceptional cases the appetite may remain relatively good. The thirst is, as a rule, not perverted, and only in occasional instances increased.

The taste is usually more or less changed; the alteration in taste is not, however, due to the gastritis alone; it is a well-known fact, for instance, that people who smoke a great deal do not eat very much, for the reason chiefly that they are usually afflicted with chronic pharyngitis or stomatitis. Stomatitis alone is sufficient to cause loss of appetite even though the functions of the stomach *per se* may be altogether intact. I think it is very doubtful whether the bad pasty taste that so many patients with chronic gastritis complain of is really due to inflammation of the stomach. If a man who smokes a great deal or drinks heavily is afflicted with chronic gastritis, there are usually at the same time some pharyngitis and stomatitis, and it is more than probable that the latter conditions can be made responsible for the bad taste in the mouth, the feeling of nausea, and similar symptoms. The bad, putrid breath of many of these patients with chronic gastritis is also usually due to changes in the mouth and pharynx, and not so much to gastritis and putrefactive processes that occur in the stomach. In all these cases the pharynx and the mouth should be carefully examined.

In some cases of chronic gastritis there is an increased secretion of saliva; in other cases, again, patients complain of abnormal dryness of the mouth. Here, too, the buccal and pharyngeal cavities should be carefully examined.

Dyspeptic symptoms proper occur with varying intensity in chronic gastritis; as a rule, they occur at the height of digestion. Some of the patients complain of an almost constant feeling of pressure and fulness in the stomach, and state that they feel bloated, etc. In other cases, again, and these constitute the majority, the above-named symptoms appear only at the height of digestion, or at least are most intense at this time, whereas in the intervals between they are insignificant or completely absent. The quantity and quality of the food exercise an important influence on the occurrence of all these symptoms. Liquid food is usually better than more solid articles of diet. Food that is easily digested causes very slight symptoms or none at all, whereas indigestible food that overtaxes the secretory powers of the stomach or some other function of the organ, causes severe distress. In general it may be said, however, that the symptoms are not very severe; the patients complain only of a feeling of discomfort or of pressure and fulness in the stomach region, a desire to belch, and similar symptoms. Only in those cases in which the tone of the stomach is very much reduced, where, consequently, the stomach retains the ingesta for an abnormal length of time, do these symptoms persist for a long period or become continuous.

Attacks of pain proper do not belong to the syndrome of ordinary chronic gastritis. It is true that the patients frequently complain of pain soon after eating, but if we examine these cases more carefully, it will be found that the sensation they complain of is not really violent pain, but merely a disagreeable feeling. This sensation is encountered with particular frequency in the atrophic forms of chronic gastritis, and the attacks occasionally recall the gastric crises of tabes. The attacks of pain and the majority of the other symptoms appear chiefly at the height of digestion; in some instances, however, they occur during the night—that is, at a time when the stomach is empty or contains nothing but a little mucus.

Many patients complain of heartburn and acid eructations. If we include under the former name only those abnormal sensations that are felt along the cardia, and are caused by the upward movements of acid masses, then heartburn proper is rarely encountered in chronic gastritis. Genuine heartburn is encountered in hyperacidity and hypersecretion; it is true that many patients complain of acid or disagreeable or putrid or tasteless belching. This symptom is produced by abnormal fermentation and decomposition that occur in the stomach, and is usually most intense at the height of digestion, although it occasionally occurs when the stomach is empty.

Many patients complain of nausea. This sensation is frequently experienced in the morning when the stomach is empty; in other cases again at the height of digestion, and in still other cases both when the stomach is empty and when it is full. Vomiting does not occur very frequently. It is quite important to determine in simple chronic gastritis whether the patient can vomit easily or whether this act is accompanied with much difficulty. Many patients are able to vomit at once as soon as they feel nauseated; consequently they proceed to vomit as soon as there is the slightest nausea, for they know that most of their distress will stop if they do this. Patients, on the other hand, who can vomit only with difficulty, suffer from nausea for a much longer time before vomiting occurs, and in many instances relief is never procured in this way.

In chronic gastritis the vomit usually consists of undigested food-remnants that remain in the stomach from the previous meal. These morsels of food are usually mixed with large or small quantities of tough mucus. If vomiting occurs when the stomach contains no food, the vomit usually consists of numerous tough masses of mucus that are occasionally mixed with bile. This appearance is presented chiefly in so-called *vomitus matutinus.* This symptom appears quite frequently in chronic gastritis; its appearance, however, does not aid us in the diagnosis of the disease. Drunkards who are afflicted with alcoholic gastritis frequently suffer from *vomitus matutinus.* Here the vomit consists of saliva that has been swallowed, and that is mixed with mucus. In some other affections of the stomach, and even in simple pharyngitis, there may occasionally be vomiting when the stomach is empty.

Other symptoms that patients with chronic gastritis complain of belong particularly to the nervous sphere; there may be vertigo and sensations of fear, particularly after eating. In other cases again the symptoms of asthma dyspepticum appear. In speaking of asthma dyspepticum we must never forget that it has nothing whatever to do with asthma nervosum. Asthma dyspepticum is merely a mild degree of dyspnea characterized by certain subjective feelings of oppression, but it does not present the well-characterized symptom-complex of genuine nervous asthma; it would be more correct, therefore, to speak only of dyspeptic dyspnea.

Other patients, again, complain of palpitation of the heart, particularly after eating, or of palpitation in the region of the stomach, of pulsations in the epigastric region, particularly at the time of digestion. All these sensations are described as very disagreeable.

The objective findings in these cases are much more important than the subjective symptoms that the patients complain of. The general health is always disturbed, even though not necessarily to any great extent, in all cases of chronic gastritis of advanced degree. The mood of the patient, above all, is very much depressed. The sufferers frequently become very hypochondriacal, and imagine that they are lost beyond hope. Every new abnormal sensation enforces this belief. They ultimately imagine that they are suffering from some very severe and altogether incurable disease. Many energetic, active men frequently become altogether incapable of doing any work and lose all their former energy. The appearance of the patient naturally is very bad. The facial expression indicates great suffering, and nutrition is also impaired whenever there is depression of this kind. In fact, the mood of these patients does as much to interfere with normal nutrition as the gastritis itself. There are, of course, a great many transitional forms between these severe cases and milder ones; in fact, many mild forms are seen in which the disease lasts for a relatively long time without marked disturbance of the general health of the patient.

We will now discuss the appearance of the tongue. Older physicians have always been in the habit of attaching great significance to the appearance of the tongue in all varieties of disease, particularly in diseases of the stomach; they imagined that the tongue was, so to say, the mirror of the stomach. While it is true that inspection of the tongue is an important adjuvant to diagnosis, no conclusions in regard to perversions of gastric function can ever be drawn from abnormalities that are seen in the tongue. It is true that the tongue is frequently covered with gray or grayish-yellow mucus, but in many cases of pronounced chronic gastritis the tongue may be free from all deposit and perfectly clean. A coated tongue is particularly common in gastritis when it occurs in drunkards or in men who smoke to excess. In these cases, however, the coating of the tongue is not due to the gastritis, but to the stomatitis and pharyngitis that exist at the same time. A thick grayish-white coat is often seen on the posterior portions of the organ, whereas the tip and the margins often remain free; the imprint of the teeth may even be distinguished at the margin of the tongue. This appearance is presented not only in gastritis, but in a great variety of other diseases of the stomach, and also, as we have said, in simple stomatitis and pharyngitis. We may say, therefore, that the appearance of the tongue is in no way characteristic for gastritis.

An increased flow of saliva is occasionally seen. This symptom is not constant, however, and in many instances the buccal cavity will be found very dry.

Fœtor ex ore, as we have mentioned, is a frequent symptom. It may be present only at certain times, especially at the height of digestion, or it may be constantly present. In the first instance it is probably due to abnormal fermentation- and putrefaction-products that are formed in the stomach during digestion. In the second case we must assume that the *fetor* is due to some disease of the mouth or pharynx. In every case of *fœtor ex ore* the last-named organs should be carefully examined.

The greatest importance, of course, attaches to the examination of the diseased stomach. This examination should include a determination not only of the size, the form, the position, and the presence of painful areas over the stomach, but also of the functional powers of the organ. The only way in which we can draw conclusions in regard to the nature of the primary disease is to determine the character of any functional perversions that may be present. My reason for emphasizing this point particularly is that this method of examination—that is, the determination of disturbances of physiologic function—is not always carried out with proper thoroughness. In milder forms of so-called chronic catarrh of the stomach many physicians consider it altogether superfluous to analyze the stomach-contents. A physician who omits this method of examination is groping in the dark. For a long time clinicians simply determined the secretion of hydrochloric acid; but this is not enough, for the hydrochloric acid question is only one of many that must be answered when the stomach-contents are aspirated for diagnostic purposes. I consider the macroscopic inspection of the stomach-contents as important as the solution of the hydrochloric acid question—perhaps more important. Direct inspection of the ingesta that are pumped out frequently teaches us at once what work the stomach is capable of performing—that is, whether the stomach is digesting the food thoroughly or not. At the same time we frequently obtain a very good idea of the motor powers of the organ. It is unnecessary to enter into details of these questions in this place. In chronic gastritis the examination of stomach-contents for diagnostic purposes is particularly desirable, for the reason chiefly that this disease is characterized by such vague and indefinite symptoms, and because it may be present in so many different degrees of severity.

The first step in the examination of the stomach should be inspection and palpation of the gastric region. In the majority of cases inspection reveals nothing abnormal. The gastric region rarely protrudes; only occasionally, when there is great development of gas, do we see a slight distention of this part of the abdomen; the same may also be seen in atony and ectasy complicating gastritis. In determining this point the time at which the examination is carried out must always be considered. If the gastric region protrudes early in the morning when the stomach should be empty, this signifies something else than if the stomach protrudes at the height of digestion. The physician should examine sufferers with chronic diseases of the stomach at different times—that is, both when the stomach is empty and at the height of digestion.

As a rule, inflation of the stomach is superfluous; the exact determination of the size of the organ is less important than the determination of its motor powers. The best way to determine the latter is, of course, to aspirate the stomach-contents and to analyze them. If there are symptoms of stenosis of the pylorus, particularly if a thickening in the pyloric region can be felt, inflation may be necessary. This procedure may occasionally show whether or not the tumor appertains to the stomach, or, better, the pyloric region of the stomach; whether the pylorus is freely movable, or whether adhesions exist.

Palpation usually discloses a certain sensitiveness to pressure, and occasionally pain. This sensitiveness to pressure is not limited to any one circumscribed area, but is usually present diffusely over the whole region of the stomach. The resistance of the organ is rarely increased; in those cases in which there is great hypertrophy of the pyloric musculature a resistant portion of the stomach may occasionally be felt in patients whose abdominal walls are very thin. If in cases of this character ectasy has developed as a result of stenosis of the pylorus, the well-known signs of the former condition, namely, succussion-sounds over an abnormally large area, peristaltic unrest of the stomach, etc., will be noticed. I will not enter into a detailed discussion of these symptoms of stenotic ectasy in this place, but refer to the sections on Ectasy and Atony.

We might expect that in the cases of so-called *cirrhosis ventriculi* that are occasionally observed the thickening of the walls might be felt through the abdominal walls. The stomach in these cases would naturally be smaller than normal. I do not believe, however, that even under these circumstances the diagnosis of cirrhosis or of cirrhotic gastritis could be made with certainty, for diffuse carcinoma would create the same picture.

The examination of the secretory powers of the stomach gives us much valuable information. In order to determine what the secretory powers of the stomach are, either the vomit may be examined or the stomach-contents may be aspirated after a test-breakfast or a test-meal.

In gastritis the quantity of stomach-contents that can be pumped out is usually larger than normal. Occasionally, however, this is not the case. A more characteristic feature of severe forms of gastritis is the appearance of the vomit; it usually contains large, coarse morsels of food that are only very little digested—in fact, they look as if they had just been swallowed. In addition there will be abundant quantities of mucus that are intimately mixed with the food-remnants. The stomach-contents are, therefore, thick, tough, and sticky. It may be difficult to aspirate these tough masses through the sound, and they are very hard to filter. The presence of mucus in the stomach-contents has for a long time been considered one of the most valuable diagnostic features. I consider this question of such importance that I may be allowed to insert a few remarks on the secretion of mucus in this place.

In order to determine whether or not there is an abnormal formation of mucus in the stomach we may adopt one of two methods: we may either examine the vomit or aspirate the stomach-contents and analyze them. The vomit is less suitable for this test, because vomiting never evacuates all the mucus, or at least does not remove it so thoroughly as it can be removed by lavage. Any physician who has performed lavage in many patients, or has washed out the stomach many times for therapeutic purposes, knows that large quantities of mucus may remain behind for a long time, so that all the food may be removed and the wash-water may run perfectly clear for a time before mucus appears.

There is one danger in diagnosing an increased secretion of mucus in the stomach, namely, that the mucus removed from the stomach may easily be confounded with mucus that has been swallowed. The presence of mucus in the stomach does not demonstrate that it has been formed there, any more than the presence of blood directly indicates gastric hemorrhage, for we know that blood that is swallowed or that comes from the esophagus may enter the stomach and simulate gastric hemorrhage.

The mucus produced within the stomach is always intimately mixed with food-remnants; it is always adherent to morsels of food, and never appears in the form of isolated balls. Only in case the stomach is washed out when it contains no food, particularly early in the morning, or if the stomach-contents is examined toward the end of a prolonged lavage, will isolated flakes and shreds of mucus occasionally be seen in gastritis. These flakes are torn from the stomach-wall by the stream of water that pours out of the sound, and soon form a sediment at the bottom of the vessel in which the wash-water is received. Mucus that does not come from the stomach, but from the mouth, the pharynx, or the larynx, presents a different appearance. This mucus is never intimately mixed with food-remnants, but always appears in the form of isolated balls that swim on the surface of the wash-water; it looks glassy and purulent, and is very frequently foamy.

We see, therefore, that macroscopic inspection alone may frequently aid us in determining whether the mucus coming from the stomach was secreted in the stomach or was swallowed.

There is another point that I must mention briefly. One might be inclined to deny that the determination of the formation of mucus in the stomach is of diagnostic value, because we know that the normal stomach secretes a certain amount of mucus. As a matter of fact, some of the older authors state definitely that washing out the stomach frequently brings up considerable quantities of mucus even in perfectly healthy subjects. This statement is unquestionably wrong; these investigators were either dealing with cases in which there was some slight abnormality of gastric function, or they mistook the mucus that they washed out for stomach mucus, whereas in reality it was probably derived from some other source. The character of the food also exercises some influence on the production of mucus in the stomach. Schüle[1] has called attention to the fact that the secretion of mucin is particularly abundant if the diet consists largely of amylaceous material. According to Heidenhain,[2] carnivorous animals secrete less mucus than herbivorous animals. It may be said axiomatically, however, that the normal gastric mucosa secretes very little mucus after a test-breakfast or a test-meal, and that it is usually impossible to find even isolated flakes of mucus in the wash-water from a normal stomach.

If there is chronic gastritis, however, large or small quantities of mucus are occasionally found in the wash-water when the stomach is washed out early in the morning before breakfast. We must consider this finding diagnostic for chronic gastritis if it can be positively demonstrated that the mucus is really produced in the stomach; in case of doubt, therefore, it is advisable to wash out the stomach not only after a test-meal or a test-breakfast, but also when the stomach is empty in the morning, and to examine the wash-water for mucus.

The chemical tests for mucus in the stomach-contents are insufficient and unimportant chiefly for the reason that the presence of mucus alone demonstrates nothing unless it can, at the same time, be determined whence this mucus comes. A chemical examination cannot, of course, determine the latter point. For all these reasons the macroscopic examination of the stomach-contents, and the discovery of mucus by this means, is the safest method, and the only one that allows us to formulate a judgment in regard to the quantity of mucus secreted by the stomach.

The majority of authors claim that the mucus in the stomach is not acted upon by gastric juice. This statement has been combated by Schmidt,[3] who has contributed much to our knowledge of the secretion of mucus in the stomach. Schmidt found that twice as much time was required to digest a given quantity of tough mucus as to digest an equal amount of albumin; but even hydrochloric acid solutions alone, according to the statements of this author, are capable of digesting mucus, although not so rapidly as gastric juice.

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[1] *Zeitschr. f. klin. Med.*, vol. xxviii., p. 481.

[2] Hermann's *Physiologie*, vol. v., Nos. 1, 2, p. 122.

[3] *Deutsch. Arch. f. klin. Med.*, vol. lvii., p. 72.

We have mentioned that gastric mucus may appear either in the form of glassy, swollen lumps, or in the form of fine flakes and shreds. This varying appearance of the mucus is due to the varying amounts of hydrochloric acid that are found in the gastric contents. Where there is lack of hydrochloric acid, the gastric mucus swells. This has been shown by the investigations of Schmidt, who expresses himself as follows: "In general we may say that the secretion of acid in the stomach is deficient wherever we find gastric mucus swollen and glassy like the mucus of sputum" (Schmidt). This shows that swollen mucus is not the only important form, although, as a rule, this form alone has been considered in determining whether or not there is an increased secretion of mucus. Much will depend on the amount of acid present. Even if the hydrochloric acid in the stomach is normal, or actually excessive, the mucus may be increased; but in these cases it will appear in the form of shreds, flakes, or fibers. In general we may say that the quantity of mucus is inversely proportionate to the quantity of hydrochloric acid secreted. The largest quantities of mucus are found where there is complete absence of hydrochloric acid. The explanation of this peculiar relation between mucus and acid in the stomach is not yet determined. Schmidt believes that it is due to the difference in the reaction of the epithelium of the stomach and of that of the glandular cells to the same irritant. I believe that certain noxious agencies that inhibit the function of the glandular cells affect at the same time the epithelial cells in the sense of causing an increased secretion of mucus. This would correspond with a statement that Schmidt[1] made some time ago, namely, that the epithelial cells are especially resistant. If this is true, we can understand why large quantities of tough mucus might be secreted even though there were a complete absence of hydrochloric acid and pepsin—as, for instance, in atrophy of the glands.

In doubtful cases a microscopic examination may be made. We may sometimes be able to determine from the presence of pigmented alveolar epithelium that the mucus comes from the air-passages; from the presence of squamous epithelium that it comes from the mouth or the pharynx. Microscopic examination of pathologic gastric mucus rarely yields much information. In general, as Jaworski[2] has shown, nothing but cell nuclei are found in it if the gastric juice is normal, whereas we find whole cells if the powers of the gastric juice are deficient. The presence of leukocytes in the stomach-contents that are aspirated when the stomach is empty is also noteworthy; other elements of the gastric mucosa are rarely found. No diagnostic or pathogenic importance can be attached to the so-called snail or spiral cells that Jaworski has frequently found in gastric mucus. A large variety of micro-organisms are, of course, always found in gastric mucus.

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[1] *Virchow's Arch.*, 1896, vol. cxliii., No. 3.

[2] *Centralbl. f. klin. Med.*, 1886, No. 49.

My reason for entering into a detailed discussion of the significance of mucus, the method of determining its presence, and the different conditions under which it is found, is that the average practitioner never pays much attention to the various conditions that I have called attention to, but usually speaks merely of gastric mucus. I am thoroughly convinced that mucus that comes from other organs and that is swallowed is very frequently mistaken for stomach mucus. The only mucus, of course, that has any pathologic significance whatever is that which is produced within the stomach. The different appearance of the mucus is also of some practical significance, for it tells us whether the gastric juice is acid or subacid.

It remains to discuss the chemical properties of gastric juice in chronic gastritis. In the majority of cases the secretion of gastric juice is more or less decreased. In cases where there is complete atrophy no gastric juice is secreted. As a rule, the reactions for free hydrochloric acid are very feeble or completely absent ; the degree of the deficit may easily be determined by adding one-tenth normal hydrochloric acid by titration ; it will vary according to the intensity of the pathologic processes going on in the mucosa.

I might mention in this place, however, that in these forms of gastritis relatively large fluctuations of the total acidity are frequently encountered ; occasionally we may observe a complete absence of free hydrochloric acid, and then the reappearance of the acid after a short time. It is not correct, therefore, to assume immediately that severe changes of the gastric mucosa have occurred if we analyze the stomach-contents only once and happen to find an absence of free hydrochloric acid. The only way in which to obtain a true picture of the secretory conditions existing within the stomach is to perform repeated examinations.

Formerly the opinion was generally prevalent that in chronic gastritis there was always an abundant formation of mucus and at the same time a great reduction in the secretion of gastric juice. Of late years, however, a special form of gastritis, called " acid catarrh of the stomach," or " gastritis acida " or " hyperpeptica," has been described. Opinions in regard to the prevalence of this form vary greatly ; some authors in describing chronic gastritis limit themselves to a description of the ordinary form, in which there is a decrease of peptic power and an abundant formation of mucus. They recognize this form alone, and do not concede that there is another form in which the production of hydrochloric acid is increased. Others again consider gastritis acida as a frequent occurrence, and still others look upon it as a rare form. There is no doubt, in my opinion, that many of the cases that are described as " acid catarrh " are really cases of chronic hypersecretion.

I believe that there are unquestionably many cases of chronic dyspepsia that may be looked upon as gastritis acida in the above sense. Cases of this kind have been described by Boas[1] in particular. In all of them there was great increase in the secretion of mucus, and at the same time an increase in the hydrochloric acid. These forms cannot, however, be regarded as frequent; many experienced observers go so far as to deny their occurrence altogether. Dyspeptic disturbances analogous to those seen in ordinary gastritis can undoubtedly also occur in simple hyperchlorhydria; nevertheless we are not justified in the latter case in speaking of gastritis acida. In order to diagnose gastritis there must always be an increased production of mucus.

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[1] *Bericht d. 66. Versamml. deutscher. Naturforscher u. Aerzte zu Wien*, 1893 ; see also *Berlin. klin. Wochenschr.*, 1894, No. 4.

We are justified, therefore, in speaking of gastritis only in those cases in which the above-mentioned symptoms appear, and there are at the same time an increase in the secretion of mucus and an increased acidity due to hydrochloric acid. But, again, as we have stated, the mere demonstration of mucus in the stomach is not conclusive, for it must first be determined whether or not this mucus is really formed in the stomach. I fear that many authors have not exercised sufficient care in considering this matter. A perusal of many of their descriptions does not inform us whether or not all these typical features have really been considered with sufficient care. In hyperchlorhydria or hypersecretion large or small quantities of mucus are frequently found in the stomach after fasting, but direct inspection readily shows that the mucus consists of thick balls and lumps, so that we are dealing not with stomach mucus, but with mucus that has been swallowed. As hyperchlorhydria is quite frequent in drunkards and in men who smoke a great deal, it is necessary to exercise particular care in interpreting the occurrence of mucus in the stomach-contents, for here it is especially easy to confuse this mucus with mucus derived from the upper air-passages.

Boas[1] deserves great credit in having first called attention to gastritis acida. In order to illustrate the disease-picture he describes in his well-known text-book on diagnosis, he reports two cases. In one of the cases the acidity was 43, in the other 56. Neither of these values can be considered particularly high. This agrees with my own experience, for I have rarely observed very high acidity in gastritis acida. As a rule, the cases are suffering from hyperacidity that is only slightly above the normal values; this, in itself, demonstrates that we are dealing with relatively slight degrees of chronic gastritis or with cases of chronic gastritis in its initial stages. It may be that some clinicians see more of these cases among their clinical material, and that, consequently, some declare gastritis acida to be more common than others. Hospital clinics, as a rule, receive more old, chronic cases than polyclinics, and in private practice more cases are seen in relatively early stages than in hospital practice. If we are correct in assuming that many of the cases described are really incipient or relatively mild forms, then the difference in the various reports might be explained on this basis. However we attempt to explain this, the fact remains that in the great majority of cases of chronic gastritis the secretion of gastric juice is more or less reduced, and that as the disease progresses, the secretion falls lower and lower. [A good deal has been said in recent literature on the occurrence of chronic acid gastritis, with the result that more importance is ascribed to the subject than it really deserves. As the author says, it is rare to find free hydrochloric acid increased in any case of gastritis, and there is only occasionally a moderate increase in the early stages of the affection. The statement that in chronic gastritis the secretion of the stomach both as to acid and enzymes decreases in proportion to the intensity and the duration of the disease should not be obscured.—Ed.] The secretion of pepsin is also reduced in severe cases of chronic gastritis. In advanced cases of atrophy not only hydrochloric acid, but also the gastric ferments, may be completely absent. Organic acids are either completely absent or present only in small quantities; occasionally they are found in larger quantities, particularly in those cases in which conditions for prolonged stagnation of stomach-contents are favorable—that is, in advanced degrees of atony. Large quantities of lactic acid are hardly ever found in chronic gastritis. Acetic and butyric and other fatty acids are occasionally seen.

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[1] *Diagnostik u. Therapie d. Magenkrankheiten*, pt. ii., second edition, 1895, p. 19.

It is natural that in those cases where the secretion of hydrochloric acid and of pepsin is reduced the digestion of albumin is correspondingly impaired. Small quantities of albumin may undergo peptonization, particularly as the production of hydrochloric acid is rarely stopped altogether, and as some pepsin is usually secreted, even in advanced stages. The appearance of remnants of meat in the stomach-contents after a test-meal varies therefore. Amylolysis is less interfered with or may even be perfectly normal.

[Roth, in Ewald's laboratory, found that pepsin secretion in disease varies in different subjects nearly as much as that of HCl. This was observed in gastritis, atrophy, etc.—Ed.]

Rennet-zymogen is usually decreased in chronic gastritis, but this reduction is not parallel to the reduction in the secretion of hydrochloric acid and of pepsin. According to Boas[1] and Bouveret,[2] the quantitative determination of rennet-zymogen is an important clinical criterion for the intensity and the prognosis of the gastritic process. The method described by Jaworski[3] is useful for this purpose. In consists in introducing solutions of hydrochloric acid of different concentration into the stomach and then determining the amount of enzyme in the solution.

The occurrence of digestion and the assimilation of the food introduced are dependent not only on the properties of the gastric juice, but also on the motor power of the stomach; if the latter is undisturbed, any lack in the secretory powers of the stomach may be compensated by intestinal digestion. The absence of fermentation and putrefactive processes under these conditions can readily be explained from the fact that the ingesta remain in the stomach for so short a time.

We have mentioned that the examination of *vomitus matutinus* and of the stomach-contents aspirated after fasting is just as important as the examination of the stomach-contents after a test-meal

[1] *Deutsch. med. Wochenschr.*, 1892, No. 17. [2] *Gaz. méd. de Paris*, 1893, No. 22.
[3] *Verhandl. d. III. Cong. f. innere Med.*, 1888.

or a test-breakfast. As a rule, mucus is seen under these conditions; its appearance we have described. The reaction of the stomach-contents is usually slightly acid or neutral, and free hydrochloric acid is rarely found. When atony does not exist, remnants of food are not aspirated. We may, however, frequently find numerous formed elements, squamous epithelia, free nuclei, and occasionally fragments of glandular tissue.

Einhorn[1] observed small shreds of mucosa in the wash-water in a number of cases. He considers this a particular form of chronic catarrh, for in his cases such shreds of mucosa were regularly found. Einhorn believes that injury to the mucous membrane by the sound was completely excluded, and states positively that the patients were suffering from chronic catarrh of the stomach with erosions. All his patients complained of slight pain in the region of the stomach after eating; they were emaciated and weak, there was a decrease in the hydrochloric acid secretion, and at the same time an increase in the formation of mucus. Only in one case was there hyperchlorhydria.

The motor power of the stomach is frequently disturbed in chronic gastritis. It may be either increased or decreased. If there is hypertrophy of the gastric muscularis, the ingesta may be propelled even more rapidly than normal. In chronic gastritis the motor power of the stomach frequently remains intact even though the secretory power of the organ is more or less decreased. This constitutes a very important compensatory process, for if the ingesta enters the intestine at an early stage and before decomposition occurs, intestinal digestion can vicariously assume the functions of gastric digestion. If the stomach-contents are aspirated in cases of this kind, only very coarse and undigested particles of food will be found. They are usually so large that they can hardly pass through the sound.

In other cases again the time of digestion is prolonged—that is, enormous, coarse, and partially decomposed remnants of food are found in the stomach at a time when the organ should normally be empty. As far as we are justified in drawing any conclusions in regard to the motor powers of the stomach from the time required for the digestion of food, we may assume that, in the latter case, the motor power of the stomach is reduced. Strictly speaking, however, such a conclusion is not justified. Before the food can pass from the stomach into the intestine it must undergo certain transformations; these are brought about partly by the action of the saliva and the gastric juice, and partly by the motor functions of the stomach. In chronic gastritis both factors may be damaged: in the first place, the secretion of gastric juice is, as a rule, reduced; in the second place, the abundant secretion of mucus prevents the impregnation of the ingesta with gastric juice, so that the maceration of the food and its transformation into a fine pultaceous mass are rendered more difficult and are retarded. The motor power may at the same time be perfectly normal. In other cases, again, the swelling of the mucosa, combined with hyperplasia of the muscularis in the region of the pylorus, may render the exit of food through the pylorus more difficult—in other words, there may be a benign stenosis of the pylorus. If this is the case, the ingesta are retained for an abnormally long time. In still other cases again there is inflammatory swelling of the mucosa, owing to the distention of the stomach and the retention of food; or the inflammatory process involves the muscularis itself, so that degenerative atrophy of the muscles of the stomach-wall results. In the former case the time of digestion will be abbreviated as soon as the inflammatory changes in the mucosa improve. If the muscularis is involved in the inflammatory process, if the muscle-fibers degenerate, advanced degrees of atony and of atonic ectasy may develop, with all that they entail, namely, fermentation, decomposition of stomach-contents, etc.

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[1] *New York Med. Record*, June 23, 1894.

Moderate degrees of atony and dilatation of the stomach are quite frequently seen after severe attacks of gastritis. When the gastritis is cured, these conditions may also be improved. Severe degrees, however, usually lead to permanent dilatation of the stomach.

It is natural that the absorption of food must also suffer in the latter forms.

The bowels are usually constipated and the stools rarely normal. In exceptional cases constipation and diarrhea alternate. In very rare instances there is diarrhea. Severe flatulence caused by the development of gas from decomposing intestinal contents is frequently seen in severe forms of gastritis in which there is prolonged stagnation of the ingesta.

The urine shows no characteristic abnormalities; its quantity is occasionally decreased and its specific gravity raised. A sediment of phosphates is frequently precipitated. A decrease in the urinary secretion is seen with particular frequency in cases of advanced atony. The action of the heart and the pulse are, as a rule, normal; some patients complain of palpitation of the heart. On examination, however, it will be found that the heart action is neither too forcible nor accelerated. Some authors claim that irregularity of the pulse is occasionally found in chronic gastritis; even if this is the case, we have nothing whatever to show that there is any causal connection between gastritis and this symptom. The pulse is quite frequently found to be accelerated, but this cannot be considered characteristic in any way for chronic gastritis. The retardation of the pulse-beat that is found in a number of other diseases of the stomach is observed only exceptionally in chronic gastritis.

In conclusion, a number of nervous symptoms may be enumerated. The most important and significant of these are certain psychic symptoms. A positive statement on the part of the physician that the disease is curable frequently relieves them. I consider another symptom that is frequently complained of, so-called gastric vertigo, to be less important, although many authors consider it to be very significant. While it is true that this symptom is occasionally seen in chronic gastritis, it is so rare that it can hardly have any direct connection with the disease. Symptoms that are much more frequently complained of are a feeling of fulness in the head, insomnia, a general lack of energy, and distaste for work.

Course.—The course of this disease as implied by the name "chronic gastritis" usually extends over a prolonged period of time—occasionally over many years. The disease frequently remits and then exhibits exacerbations. The general course of the disease will naturally depend on the duration of the trouble, on the severity of the changes, on the mode of life, the diet, the primary cause, and many other factors. Mild cases that are not too far advanced can usually be cured, provided the manner of life is carefully regulated. Recurrences, however, frequently happen. Many patients, as soon as they improve a little, immediately expose themselves to the same influences that originally caused the trouble. This is seen particularly in drunkards. Even severe cases of gastritis are frequently seen to suffer for many years without showing any appreciable impairment of their general health. As a rule, these patients hold their own fairly well as long as the motor power of the stomach remains intact. We have many observations to show that the secretion of gastric juice may be greatly damaged or even completely lost, and still the assimilation and absorption of food be only slightly impaired, provided the motor power of the stomach remains good. As soon, however, as the motility of the stomach becomes seriously impaired, assimilation and absorption, and consequently the general nutrition and health of the patient, are very much impaired. Loss of motility may result, on the one hand, from a stenosis of the pylorus that secondarily follows chronic gastritis. If this occurs, the propulsion of stomach-contents into the duodenum is rendered more difficult. Another cause is direct weakening of the gastric musculature. Under these circumstances the symptoms of more or less advanced motor insufficiency are added to those of chronic gastritis, and finally advanced degrees of ectasy with all its complications—that is, abnormal fermentation, decomposition, etc.—supervene. It is, of course, not surprising that under these circumstances the nutrition of the patient suffers greatly. Any impairment of the tone of the gastric muscularis and any factor that hinders the propulsion of the ingesta exercise an unfavorable influence on the course of the disease.

Chronic gastritis rarely terminates in atrophy—in so-called achylia gastrica. We will not enter into the question whether or not atrophy of the gastric mucosa is always due to chronic gastritis or whether it can also originate from other causes. Atrophy of the gastric mucosa, at all events, is a typical clinical disease-picture that merits particular discussion.

The course of other forms of secondary gastritis that follow other chronic local or general diseases will naturally depend on the primary disease. If the latter are curable or are capable of improvement, the gastric symptoms are also improved ; if the primary disease is incurable, the gastritis will gradually progress from bad to worse.

Diagnosis.—The diagnosis of chronic gastritis is not so easy by any means as older physicians seem to believe, and as many modern physicians assume to-day. In the first place, I wish to emphasize the fact that the diagnosis "chronic gastritis" can never be positively made from the clinical symptoms alone, but only with the aid of a careful analysis of the stomach-contents. The subjective symptoms of the patients and the objective findings elicited by the ordinary methods of examination reveal nothing whatever that is characteristic. Analogous symptoms are seen in purely nervous dyspepsia, in carcinoma in its early stages, etc.

Neuroses of the stomach and carcinoma of the stomach are most frequently confounded with chronic gastritis. I will not mention so-called atrophy of the gastric mucosa, because I will discuss this disease in the following section, and will include under the term "chronic gastritis" only those forms in which the complex of dyspeptic symptoms characterized by a reduction in the secretion of gastric juice and the abundant formation of mucus that we have described above is seen. I have already mentioned that in exceptional cases there may be a slight increase of the gastric secretion together with an increase in the production of mucus.

Although ordinary chronic gastritis can produce the same dyspeptic disturbances as a neurosis of the stomach, the symptoms in the latter disease are never so uniform nor so persistent as in gastritis. As in other forms of neurosis, the disease-picture in neuroses of the stomach varies. The different disturbances are not so dependent on the quality and the quantity of food as in chronic gastritis; in the latter disease any excess in eating and drinking, the ingestion of indigestible food, invariably increase the distress of the patient. In nervous dyspepsia, on the other hand, this interrelationship between the food and the symptoms is rarely so apparent. It is true that great distress may follow an inappropriate diet in neurosis, but it does not occur as a rule. In this condition indigestible food may be well borne and a very digestible diet may cause severe symptoms; on one day the same article of food may agree very well, while on another day it will produce all manner of symptoms.

All these criteria may be insufficient in certain cases, and even a most experienced practitioner may be deceived if he attempts to build up a diagnosis from these symptoms alone. The only way in which to arrive at a positive diagnosis is to examine the stomach-contents. In this examination the determination of the hydrochloric acid is less important than the examination of the mucus. The presence of large quantities of mucus, provided it can be demonstrated that it comes from the stomach, is an absolutely positive criterion.

It is always well to wash the stomach thoroughly after aspirating the stomach-contents, for frequently abundant quantities of mucus will be found in later washings. Washing out the stomach after fasting is also a good method for detecting the presence of mucus. Penzoldt recommends introducing a little bismuth into the stomach in order to render the detection of mucus more easy. The little grains of bismuth, it seems, adhere to the mucus and make it more easily seen. The hydrochloric acid secretion is less important. It may be said that in ordinary chronic gastritis the secretion of gastric juice is, as a rule, reduced; but in exceptional cases of nervous disorders of the stomach the hydrochloric acid secretion may also be diminished. In neurosis of the stomach, it is true, the hydrochloric acidity usually fluctuates within wide boundaries; whereas in chronic gastritis the hydrochloric acid secretion is more uniform. As in all other forms of chronic disease of the stomach, the physician should make it a rule to withhold judgment in regard to the gastric secretion until he has performed a number of analyses of the stomach-contents. He should never content himself with one examination. The correspondence and the uniformity of the findings in these cases are very important in rendering a diagnosis of gastritis.

I have already mentioned that hyperchlorhydria may also occur in chronic gastritis. My personal experience seems to teach that this occurrence is rare, and that hyperchlorhydria is seen only in mild forms and in early stages of chronic gastritis. Hyperchlorhydria is also seen in neuroses of the stomach, but in the latter conditions the values for hydrochloric acid fluctuate. We are justified in declaring hyperchlorhydria a symptom of chronic gastritis only in those cases in which we observe the general symptom-complex of chronic gastritis together with an abundant production of mucus. Boas correctly attaches much importance to the enzymes of the stomach-contents. In advanced degrees of chronic gastritis the latter are always found decreased, whereas in mild forms and in early stages of chronic gastritis they will be present in normal quantities. In neuroses, to judge from my experience, the enzymes are usually present in normal amounts.

In isolated cases small shreds of the mucosa may occasionally be found in the wash-water that comes from the stomach. I am hardly inclined to attach much importance to the microscopic examination of these shreds for the purpose of deciding whether or not the mucous lining of the stomach is intact or inflamed. In the first place, these shreds of mucosa are rarely found. If the sound is carefully withdrawn while the water is still running, shreds of mucous membrane will rarely be torn off. And, finally, I must consider it a very precarious procedure to draw conclusions in regard to the state of the whole mucosa of the stomach from the examination of small shreds that are torn off; at least I should venture to say that even if these shreds are found to be normal, we are not justified in excluding gastritis.

Disturbances of motility that we might discover can hardly be utilized in rendering a diagnosis. The ingesta that are removed at the usual time after a test-meal usually appear quite coarse in chronic gastritis. The test-breakfast is rarely changed as it would be in a normal stomach—that is, it is not converted into a pultaceous mass. We see from this, therefore, that the food remains in the stomach longer than normal, but this is not characteristic in any way, for it may be the direct result of the decreased secretion of gastric juice, and is consequently seen in all cases where such a reduction obtains.

From all that has been said we learn that the diagnosis of chronic gastritis can be made only if all the symptoms are considered, including the results of the analysis of the stomach-contents. Only if the disease-picture in its totality and *all* the symptoms fit into the frame of chronic gastritis should we make this diagnosis.

It seems hardly possible to confuse carcinoma of the stomach with the simple form of chronic gastritis. It might be confused with the more severe form of gastritis that leads to complete disintegration of the glands, to atrophy and anadeny. In carcinoma the hydrochloric acid secretion is reduced from the very beginning, so that absence of hydrochloric acid is one of the earliest symptoms. I have repeatedly discovered complete absence of free hydrochloric acid in carcinoma at a time when no tumor could be felt, when there was no ectasy, and when there were only slight dyspeptic disturbances that might very well have been due to chronic gastritis. In cases of that character I have repeatedly felt justified in making the diagnosis carcinoma, even though hematemesis, tumor, ectasy, and cachexia were absent. My chief reason for doing this was the short duration of the disease, for in carcinoma the absence of free hydrochloric acid and the reduction or inhibition of all peptic power are early symptoms, whereas in chronic gastritis they appear late.

The form of chronic gastritis that we are discussing never leads to a complete loss of the peptic powers of the stomach, whereas carcinoma leads to such a loss very early in its course. The only exception to this rule is the form of carcinoma that develops from ulcer.

We will discuss the differential diagnosis between carcinoma and atrophy of the gastric mucosa in the following section.

The differential diagnosis between chronic gastritis and amyloid degeneration of the mucous membrane of the stomach should not present any formidable difficulties. We have found in my clinic that a reduction in the secretion of gastric juice—in fact, complete absence of free hydrochloric acid—may be encountered in amyloid degeneration, but this alone should not lead to confusion with chronic gastritis. Amyloid degeneration is not a primary disease, but a secondary one, following chronic suppuration, chronic tuberculosis of the lungs, etc. We are always, therefore, dealing with a secondary process, and the only point to be decided is whether the case is one of secondary catarrh or of secondary amyloid degeneration. The latter diagnosis should be made only if amyloid degeneration of other organs can be demonstrated. Amyloid degeneration never appears at first and alone in the gastric mucosa, but is always found in the liver, the spleen, the kidneys, etc., at the same time. In rare cases it might be assumed that analogous changes have occurred in the gastric mucosa if, together with amyloid degeneration of other organs, there should be found a constant decrease or an absence of hydrochloric acid secretion. But even under these circumstances the diagnosis could hardly be made with absolute certainty. Inversely the diagnosis can certainly be excluded if all symptoms of amyloid degeneration are absent in other organs.

I do not think it is possible to confound round ulcer of the stomach with chronic gastritis if all the features of the syndrome of the latter condition are carefully weighed. In cases of chronic gastritis complicated with a mild degree of hyperchlorhydria one might think of the possibility of an ulcer; but the picture of chronic gastritis in these cases often has no resemblance to that of ulcer. There is never cardialgia nor hematemesis, nor are there any pressure-points. I am inclined to attach particular importance to the mucus. Many authors make the statement that chronic gastritis frequently accompanies ulcer of the stomach; this I do not consider correct. In cases of typical ulcer the secretion of mucus is never increased; if we find an increase of mucus, there is always some complication; if there is an abundant production of mucus in the stomach, the impregnation of the ingesta with gastric juice is impeded by the thick layer of mucus that covers the surface of the gastric mucosa. In ulcer, unless it is complicated with some other disease, we usually find that digestion is very good—in fact, occasionally abnormally rapid. The most important symptoms, therefore, of simple chronic gastritis, namely, reduced secretion of gastric juice, abundant formation of mucus, slow or difficult digestion of albumin, are never seen in ulcer.

Prognosis.—Very little need be said in regard to the prognosis. Mild degrees of chronic gastritis, provided they have not existed too long, may be cured and frequently are cured. The fundamental condition for this favorable outcome is, of course, that the noxious agency that primarily caused the disease ceases to act. If severe degrees of atony have developed, the prognosis is less favorable; as a rule, however, atony is not very considerable, and may recede together with the other symptoms under proper treatment and a suitable diet. If there is hypertrophy of the muscularis and stenosis of the pylorus, the prognosis is less favorable, and it is still worse if there is much atrophy of the mucosa. The prognosis should not be made from the general condition of the patient, as this may be very poor even in mild cases of gastritis, but should be made exclusively from the condition of the gastric juice, of the hydrochloric acid secretion, the enzyme-production, and, above all, of the motor power of the stomach. If the latter is reduced and does not improve under proper treatment, the prognosis is bad. On the other hand, complete absence of gastric secretion may be tolerated fairly well as long as the motor power of the stomach remains intact. This is due to the fact that under these conditions the small intestine vicariously assumes the function of the stomach. We learn from clinical observation that this compensatory process may be carried on for years without damage to the patient.

In the secondary forms of gastritis the prognosis naturally depends on the primary disease. If this is curable, the gastritic condition is also curable, provided it has not advanced to extended atrophy of glands. The majority of these primary diseases are not curable, so that the prospect of a complete cure of this form of gastritis is unfavorable.

The Treatment of Chronic Gastritis.—**Prophylaxis.**—Theoretically it is an easy matter to explain how the occurrence of chronic gastritis can be prevented. We should not eat too rapidly, should avoid extreme temperatures in food and drink, should masticate and insalivate the food thoroughly, should not overload the stomach, should take good care of our teeth, should have a well-fitting set of teeth adjusted if there are numerous dental defects, should avoid excessive use of alcohol, particularly in concentrated forms, should not smoke too much, particularly strong cigars, etc.; in other words, we should avoid all the above-named things that we know from experience may lead to the development of chronic gastritis. As a rule, however, physicians are not consulted in regard to what had best be done in order to keep the stomach healthy, but in order to help after the stomach is diseased. Occasionally a physician may be able to give prophylactic advice if a patient comes to him with acute gastritis. In all these cases he should prescribe a strict diet and a sensible mode of life after recovery.

We might expect that the occurrence of secondary gastric disturbance could be prevented in all those diseases that can produce secondary dyspeptic disturbance, as, for instance, tuberculosis, emphysema, diseases of the heart, the liver, and the kidneys, and certain metabolic and nutritive disturbances. I will not enter into a discussion of the theory held by many clinicians that the dyspeptic disturbances seen in these diseases are identical with chronic gastritis. We can hardly speak of a prophylaxis in these cases, for every physician will naturally attempt to cure these diseases as well as possible, or to institute measures that are intended to compensate any perversions of function that may exist. If we made it a rule in diseases of the heart, for instance, to institute treatment as soon as compensation begins to fail, and not after it has failed, we would, in many cases, be able to prevent so-called catarrh of the stomach from stasis. In this sense digitalis may be called a *stomachic* in diseases of the heart, and in this sense only can we speak of a prophylaxis of gastritis. Strictly speaking, however, this is not a special prophylaxis against gastritis, but only one of the measures that are employed to prevent the extension of a primary disease that involves not only the stomach but numerous other organs also. The careful dietary regulations that we prescribe in diseases of the kidneys, the heart, the liver, etc., are not altogether for the sake of preventing secondary gastritis that may complicate these diseases, but for the primary affections themselves.

Treatment Proper.—We have three means at our disposal for treating chronic gastritis, viz., mechanical, dietetic, and medicamentous methods.

Among the mechanical methods of treatment lavage of the stomach occupies first place. As I have already stated in the symptomatology, lavage of the stomach should always be performed in chronic gastritis if for no other purpose than to aid in determining the diagnosis. Washing out the stomach once, however, is never sufficient; the stomach-contents should be analyzed a number of times, and, in order to verify the results, the analysis should be repeated from time to time. This diagnostic procedure alone will accustom the patient to the introduction of the stomach-tube. After a few aspirations the patient will be so well accustomed to the sound that he will not object to its employment for prophylactic purposes.

I believe that the stomach-tube is one of the most valuable diagnostic adjuvants in the treatment of chronic gastritis complicated by increased secretion of mucus. In many cases, it is true, the stomach-pump can be dispensed with, and many cases undoubtedly recover without its employment. Whether the cure of these latter cases would not have been accelerated had the sound been employed, whether many of the cases that never were cured would not have been cured had the sound been used, is necessarily an unanswerable question. I am decidedly of the opinion that these cases would have been better had the stomach-sound been employed. In complete atrophy of the gastric mucosa the sound is unnecessary, provided the motility of the stomach is intact; we can also do without it in mild forms of gastritis. Wherever there is abundant secretion of mucus the employment of the sound is indicated. There is but one way to remove large quantities of mucus, and that is through the sound.

In the general part of this work I have formulated a general rule in regard to therapeutic lavage, viz., that the ingesta should never be removed from the stomach unless they are still present in the organ after the expiration of the normal period of digestion, provided, of course, that a diet is given that is suitable to the individual case. If a certain quantity of food is still present in the stomach seven hours after a simple midday meal, this old, and frequently decomposed, food residue should be removed before new food is introduced, otherwise the stomach will become overloaded, and the new ingesta will immediately undergo abnormal fermentation and decomposition, and this, of course, would interfere with assimilation.

But the mucus itself is removed best by lavage. As a rule, only relatively small quantities of mucus are evacuated with the food, but if the stomach is thoroughly washed out until the wash-water runs clear, large quantities of mucus may be found in subsequent washings. Unless there is an advanced degree of atony, it is a good plan to remove these tough adherent masses of mucus by forcing water into the stomach under slight pressure—of course, only after all food remnants have been removed. The stomach should be washed out first with the patient in the erect position, then when he is lying down or in other positions of the body. Penzoldt, in addition, recommends massage of the gastric region. It is also well to add to the last wash-water remedies that can dissolve mucus, for instance, alkalis, sodium bicarbonate (one to two teaspoonfuls to a liter of water), lime-water (four to five teaspoonfuls to a liter of water), common salt (10 gm. to 1 liter). Fleiner recommends a mixture of common salt and soda, of the former 2 parts, of the latter 1. Of these mixtures he adds 1 teaspoonful to 2 or 3 liters of water at 26° R (90.5° F.). Some of the ordinary mineral waters may also be used for this after-lavage.

[A solution of boric acid in the proportion of a teaspoonful to a liter of water is one of the best preparations to employ after the stomach-contents is removed.—Ed.]

If the stomach contains many fermenting organisms, certain antifermentative remedies may be added to the wash-water. In my practice salicylic acid, 1 : 1000, has been found to be the best remedy for this purpose. Kuhn[1] performed a number of experiments in my clinic and found that this remedy is the best disinfectant against gaseous fermentation. Other remedies that have been recommended are thymol (0.5 : 1000), butyric acid (6 : 1000), resorcin (2–5 : 1000), benzol (5 : 1000), and weak solutions of hydrochloric acid (5–8 : 1000). The former remedies are prescribed best in powder-form, with the direction to add one powder to a liter of warm water.

[Potassium permanganate (0.05 : 1000) is a useful and safe antiseptic.—Ed.]

In all cases in which there is stagnation of stomach-contents I have previously recommended lavage of the stomach in the evening—that is, before supper. Here, however, where the chief indication is to remove the adherent masses of mucus, this regulation may be modified. If the ingesta remain in the stomach for an abnormal time, evening lavage is indicated as in any other case, particularly if there is much decomposition and fermentation, or if there is much atony of the stomach. In order to remove the mucus, however, it is best to perform lavage in the morning before breakfast, for at this time the removal of the mucus is most easily accomplished. If the mucosa is completely atrophied, but the motility of the stomach preserved, methodic lavage is unnecessary. If some authors speak of catarrh without formation of mucus and advise against performing methodic lavage in these cases, I might remark that in cases of this kind there is really no catarrh, or, better, no gastritis. Neither the dyspeptic symptoms nor the decrease in the gastric secretion allows us to make the diagnosis of gastritis. In order to be able to say that we are dealing with a case of gastritis, there must always be an increased secretion of mucus. The fact that this increased secretion cannot always be demonstrated does not change its significance. We may expect *a priori* that there will be certain differences in the degree of this secretion. In milder cases methodic lavage may be dispensed with or may be replaced by other measures; in advanced degrees, it appears to me it is the most suitable remedy. I will never concede, however, that we are justified in distinguishing between catarrhs with and catarrhs without formation of mucus. If, in advanced cases of atrophy the epithelium that furnishes mucus ultimately perishes, we are then no longer dealing with gastritis, but with a sequel of this condition, namely, complete atrophy of the gastric mucosa.

The frequency with which lavage should be performed naturally depends on the stage of the gastritis. In the majority of cases lavage once a day is sufficient. In obstinate cases with abundant formation of mucus and severe degrees of atony lavage may be performed twice a day; once in the morning before breakfast and once in the evening before supper.

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[1] *Zeitschr. f. klin. Med.*, vol. xxi.

The results of this mechanical method of treatment are frequently startling in cases of gastritis that are not too old. The patients frequently feel much better after a very short time; after a few treatments the appetite increases and all other symptoms decrease in severity. In advanced and old cases this method of treatment must, of course, be carried out persistently for some time, but here, too, the impression is created as though lavage were followed by marked benefit.

The internal stomach douche may frequently be employed with profit in chronic gastritis. This mode of treatment is particularly indicated in cases of mild atony, for here it stimulates the tone of the gastric mucosa. The water used for the stomach douche should, as a rule, be a little colder than the water used for ordinary lavage. It may also be useful under certain circumstances to force it into the stomach under pressure. In order to stimulate the secretion of gastric juice a little salt (a teaspoonful to a liter of water) may be added to the irrigating fluid. I need hardly mention that the stomach douche should only be used when the stomach is empty. Fleiner recommends washing the stomach thoroughly and then irrigating its walls with certain bitters in order to stimulate the appetite. He advises, for instance, decoctions of hops or of quassia wood, or a solution of a teaspoonful of the fluid extract of condurango in a liter of water.

If there is much atony or ectasy, the stomach douche should be employed with care. If there is complete atrophy of the gastric mucosa, this method of treatment is, of course, utterly useless.

Electricity, massage, and hydrotherapeutic procedures are generally not called for in ordinary gastritis. The electric current might be used with profit in cases where there are severe symptoms of sensory irritation or where there is advanced atony. In chronic gastritis there is usually no reason why electricity should be employed, nor is there any call for massage unless it is instituted for a coexistent atony. Hydrotherapeutic procedures might be indicated by some special conditions in individual cases. Chronic gastritis *per se*, however, rarely calls for their employment. In very sensitive subjects moist felt sponges or Priessnitz compresses may be used in order to promote a uniform distribution of heat.

The Diet.—The diet is more important than the employment of physical methods. In chronic gastritis, however, it is exceedingly difficult to formulate any definite rules in regard to the diet, to arrange any uniform lists that would be applicable to all cases. This is due to the fact that chronic gastritis, like other diseases of the stomach, occurs in so many different forms. As a rule, the secretion of gastric juice is more or less reduced, and increased only in exceptional cases. The motor power is usually well preserved and rarely disturbed to any great degree. The quantity and quality of the food must, of course, vary according to the state of the secretory and motor functions of the stomach.

The general rule can be formulated that the diet should be as digestible as possible. The term digestible is, of course, relative; in the case under discussion, it signifies a diet that makes small demands on the secretory powers of the stomach, that considers the reduction in the secretion of gastric juice, that does not irritate the gastric mucosa, and that can be propelled from the stomach in a relatively short time. If all these postulates are fulfilled, the food will not remain in the stomach for a long time, and those portions of it that are not digested will be propelled into the intestine within so short a time that they cannot undergo decomposition. If food is administered on these principles, the muscles of the stomach are not overtaxed, nor is the mucous membrane irritated; the food, moreover, enters the intestine very rapidly. The first condition of digestibility, therefore, in these cases, is that the food be administered in a suitable form—*i. e.*, should be finely distributed and preferably be given in the form of mushes or liquids.

In regard to the quality of the food, we may say that there is no reason why albumin and carbohydrates and fats should not all be given. One might imagine that the quantity of albumin should be reduced because the secretion of gastric juice is, as a rule, more or less reduced. This, however, is only partly true. No doubt the peptonization of proteids suffers when the secretion of gastric juice is reduced, whereas ptyalin action remains undisturbed. It might appear, therefore, that an amylaceous diet would be more appropriate than a meat diet; experience has taught us, however, that the assimilation of albumin may occur in a perfectly normal manner, even though the stomach has lost all its peptic powers, provided only that the motor power of the organ is intact, so that the intestine can vicariously assume the functions of the stomach. As long, therefore, as the motor power of the stomach is intact moderate amounts of albumin are permissible. The only way to decide just how much meat may be allowed is to examine the stomach-contents repeatedly; this gives an exact index of the state of the peptic powers of the stomach, it also informs us whether or not the stomach is capable of propelling the ingesta into the intestine within a normal time and before decomposition has occurred.

In cases in which the motor power of the stomach is reduced, the quantity of albumin must naturally be limited. Proteids should under all circumstances be given in a finely divided state, preferably in a readily soluble form. They may occasionally be given as peptones, albumoses, somatose, etc. A very good substitute for meat that I can recommend is nutrose ("casein-natrium").

There is no objection to the administration of carbohydrates provided they are given in a suitable form. The only articles of food belonging to this class that should be excluded are those that contain much cellulose and those that readily undergo fermentation.

There is, further, no objection to the administration of fat. Very much, of course, will depend on the form in which it is given. In all cases where nutrition is much reduced an abundant quantity of fat should be given. Fat possesses a high caloric value, and is consequently a very valuable means to improve nutrition.

In general, therefore, the diet should be mixed. It should be finely divided, pultaceous or fluid, should contain much carbohydrate, but also moderate quantities of albumin and of fat. Many variations are naturally permissible, and the exact dietary will depend on the degree of secretory perversion, the motor powers of the stomach, and the general nutrition of the patient.

A few words may be permitted in regard to the individual articles of diet. Among the cereals I might mention Zwieback, toast, Hundhausen's aleuronat flour that Ebstein recommends, Gericke's "Kraftzwieback," and "Kraft-bread." Cereals may also be given in the form of soups. Oatmeal is very nourishing and proper for these cases. Among leguminous articles of food Hartenstein's leguminose, Knorr's different preparations of leguminose, and Liebig's maltoleguminose may be mentioned. All these preparations are rich in carbohydrates and contain a relatively large amount of proteid. Potatoes should only be given mashed. Mashed carrots, spinach, soufflets, different puddings, like tapioca, etc., may all be given for the sake of variety.

Among meats, the digestible varieties (compare the scales given on page 197) should be preferred; for instance, calves' thymus, scraped beefsteak, pigeon, chicken, beef-hash, scraped raw ham, and fish that is not too fat. The quantity of meat given at one time should never be large, rarely more than 100 to 150 gm.

It will always be necessary to experiment with milk, as it is impossible to say in advance whether or not it will be well borne. If rennet-zymogen is absent, fermentation easily occurs. If the secretion of rennet-ferment is reduced, but the rennet-zymogen is still present, the addition of a little lime-water (one to two tablespoonfuls to a quarter of a liter of milk) will render the milk more digestible. Many people still labor under the impression that patients with diseases of the stomach should eat as little fat as possible. Von Noorden deserves the credit of having shown that in many cases of stomach trouble the ingestion even of large quantities of fat is not only not harmful, but, on the contrary, very beneficial. Very much, of course, will depend on the form in which fat is taken. It is best given as fresh good butter or as cream.

A few words in this place in regard to a course of mineral waters that is so frequently advised in chronic gastritis. If such a course is undertaken, certain rules and regulations in regard to the diet should be observed. In many watering-places the patients undergoing the course are advised to live up to certain detailed regulations that they are led to believe should be followed if the particular water is taken. In many watering-places these dietary regulations can even be procured in printed forms that describe exactly what food and drink is permissible in ordinary quantities, what food and drink should be reduced or stopped altogether. Dapper[1] has recently shown that all such dietary formulæ are altogether irrational. The use of any particular mineral water does not render certain articles of food unsuitable nor make it imperative that all the patients should live on the same diet ; the individual disease should always determine the diet. In many of these watering-places fats in particular are excluded from the dietary.

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[1] *Zeitschr. f. klin. Med.*, vol. xxx.

Von Noorden and Dapper have shown for Kissingen and Homburg waters (and the same could probably be done for other waters) that the ingestion of large quantities of salt water in no wise contraindicates the ingestion of even abundant quantities of fat. Dapper's investigations demonstrate that large quantities of fat and of mineral waters can be taken at the same time without materially interfering with the digestion of the former.

In regard to alcoholics, we may say that strong alcoholic drinks, also beer, should be avoided, the latter chiefly on account of the fermenting organisms that it contains. Gastritis *per se* does not call for alcoholics. Alcohol may possess some power of stimulating secretion, but this is much too slight to warrant the employment of this drug for therapeutic purposes. If alcohol is indicated for any particular reason, it should be given in small quantities and preferably in the form of a pure strong wine that does not contain much tannic acid. Such wine may be diluted with some mineral water.

The patients should not drink much water, particularly during meals.

Strong spices, like mustard, pepper, etc., may occasionally be given to stimulate the appetite, but they should not be given too frequently nor in too large quantities.

No fixed rules can be formulated in regard to the frequency with which food should be taken and the quantity to be eaten at each meal. It may be said in general that it is always better to eat small meals at frequent intervals.

From all that we have said it is clear that no fixed rules can be arranged that would be suitable for all cases of chronic gastritis ; so much depends on the general strength of the patient and the state of the secretory and motor powers of the stomach that the diet must vary in each case. In carcinoma and ulcer it is much easier to arrange a uniform dietary than in the different forms of chronic gastritis. The dietary given on page 518 (Table I.) (after Wegele) may be regarded as a fair example of a diet-list that is suitable to a case of chronic gastritis in which the gastric digestion is reduced.

Other examples of dietaries appropriate to this condition will be found on pages 518, 519 (Tables II. and III.).

[In regard to the articles of food recommended in these tables, it may be said that with Americans suffering from chronic gastritis, considering the taste of the patient and at the same time the requirements of the physician, it would be better to omit oatmeal altogether ; barley is admissible in soup only when the grains are thoroughly crushed ; scraped beef is preferable to scraped ham, but beef very thoroughly stewed or braised, by which means the muscle-fibers are well disintegrated, is still better. Veal should be interdicted with most patients.

Carrots and similar roots are best avoided. Certain vegetables that are not to be generally recommended are found to be suitable for certain patients, although a source of distress to others. The vegetable list is best formed after trial, and estimating the results by the sensations of the patient, and especially by studying the gastric contents after lavage. —Ed.]

I.—Dietary for Chronic Catarrh with Reduced Gastric Digestion.

	Albumin.	Fat.	Carbohydrate.	Alcohol.
In the morning:				
150 gm. of peptone cocoa	8.00	6.0	7.50	
25 gm. of butter (on a toasted roll)	0.18	20.8	0.15	
In the forenoon:				
Soft-boiled egg	6.0	5.0		
At noon:				
200 gm. of oatmeal soup	12.50	0.3	18.0	
150 gm. of poultry	28.0	13.5	1.8	
200 gm. of carrots	2.14	0.4	16.3	
In the afternoon:				
150 gm. of peptone cocoa	8.0	6.0	7.5	
25 gm. of butter	0.18	20.8	0.15	
In the evening:				
200 gm. of barley soup	3.2	6.0	17.0	
One egg	6.0	5.0		
Scraped ham	25.0	8.0		
100 gm. of macaroni	9.0	0.3	76.7	
In the course of the day:				
200 gm. of wine	. .	. .	6.0	16.0
75 gm. of Zwieback	9.0	1.5	63.9	
	117.2	94.6	236.01	190
Total	480	890	970	100
	2440 Calories.			

II.

	Albumin.	Fat.	Carbohydrate.	Calories.
In the morning:				
500 gm. of milk, 3 Zwieback	20.6	20.2	45.7	461
In the forenoon at 10 o'clock:				
Oatmeal soup, with yolk of 1 egg	5.3	5.2	14.2	129
At noon:				
Rice soup, with yolk of 1 egg	4.5	9.2	15.2	167
English beefsteak, 200 gm.	42.8	10.4	. .	272
25 gm. of toast	2.0	0.2	19.0	90
100 gm. of mashed potatoes	2.2	5.1	17.0	125
In the afternoon:				
250 gm. of milk-cocoa, 3 Zwieback with jam	13.5	15.8	44.6	385
In the evening:				
Rice-flour mush with 500 c.c. of milk and 30 gm. of sugar	24.4	18.8	130.8	812
25 gm. of toast	2.2	0.2	19.0	90
Total calories				2531

In regard to the further details, I refer particularly to the text-book of Biedert, and to Langermann's[1] *Dietetics and Cook Book for Patients with Gastric or Intestinal Diseases.* In this book numerous dietaries are given, and the caloric value of the different articles of food is also calculated; besides there are careful rules for preparing the different articles of food.

III.

	Albumin.	Fat.	Carbohydrate.	Calories.
In the morning:				
Milk-cocoa made from 20 gm. of cocoa, 10 gm. of sugar, and 250 c.c. of milk	10.3	18.2	25.7	321
In the forenoon at 10 o'clock:				
50 gm. of toasted wheat bread	4.3	0.5	39.0	160
100 gm. of breast of young chicken (weighed raw)	19.6	2.8	. .	106
30 gm. of butter	. .	23.0	. .	214
At noon:				
Potato soup (made from 100 gm. of potatoes, 50 c.c. of milk, 5 gm. of flour, 5 gm. of butter)	3.5	6.0	23.1	165
Veal, hashed (200 gm. raw)	42.8	10.4	.	272
150 gm. macaroni (50 gm. unboiled)	4.2	6.4	38.0	232
In the evening:				
Soup made from tapioca flour, 1 egg, 10 gm. of butter	7	14	30	282
Later:				
250 c.c. of milk, 2 Zwieback	10.9	10.5	26.3	250
Total				2002

A third method that we can employ in treating chronic gastritis is drug-treatment. I include mineral waters in this category. We know from experience that certain mineral waters are useful in the treatment of a large variety of stomach diseases and particularly of chronic gastritis. It has never been determined exactly how they act. No exhaustive and careful investigations on the influence of different mineral waters on gastric secretion, on absorption, on the proteid metabolism, etc., have been made. So far there are only a few isolated investigations in this direction on record. In general, water-cures act very much like methodic washing of the stomach. We advise such a course of treatment where we wish to produce evacuation of stomach-contents and solution of gastric mucus, where we desire to rid the stomach of stagnating contents, or where we desire to aid the evacuation of stomach-contents from the stomach into the intestine, or finally where we wish to improve the reduced glandular powers of the organ. This end can probably be attained much better and much more rapidly by methodic lavage than by giving mineral waters in large quantities. This is particularly the case if we add substances to the wash-water that dissolve mucus and disinfect the stomach-contents. In a certain number of cases, however, mineral waters may be preferable. Courses of mineral waters are always taken at some watering-place, where the patients live under a strict regime and are not exposed to a great many noxious agencies, as over-exertion, excitement, etc.; while they are there, in other words, they devote all their time to the improvement of their health. The majority of patients that are treated at home may follow the rules and regulations of the attending physician in a general way, may even undergo methodic treatment with the sound, but they hardly ever give up their ordinary occupation and mode of life. For these reasons it is very difficult to treat stomach cases at home as strictly as they should be treated. Stomach cases that are treated in a well-regulated stomach-sanitorium undoubtedly do very much better than when they are treated in any other place.

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[1] I can recommend this little book, which is intended both for physicians and patients, very warmly to those physicians who wish to gain information in regard to diet. At the same time I think it would be well if it were used only by physicians, and not by the patients themselves.

For the treatment of chronic gastritis salt waters, alkaline, alkaline-muriatic, and alkaline-saline waters are all useful. It is frequently difficult to determine which water the patient should take. It is altogether impossible to say, for instance, that in one case where the secretion of gastric juice is perverted in one way, alkaline water should be given, in another salt water, etc. We must consider all the perversions, and not only the perversion of secretion, so that advice can only be given after a careful study of the motility and absorptive powers of the stomach and the general strength of the patient. In general we may say that alkaline and alkaline-muriatic waters are indicated, particularly in those cases where we desire to dissolve abnormally large quantities of mucus or where we desire to neutralize abnormal quantities of acid. It is doubtful, on the other hand, whether these waters, as some investigators claim, can stimulate the glandular activity of the stomach. If they have this power at all, they possess it to only a very slight degree when given in small doses; if administered in large doses, they probably produce the opposite effect. Salt waters, as Kissingen, Homburg, and Wiesbaden waters, however, are known to stimulate the secretion of hydrochloric acid.

Dapper has performed some very careful investigations on patients who were undergoing a typical cure both in Kissingen and Homburg, and has shown that the prolonged use of these salt waters causes subacidity to disappear, and leads to a general improvement in the patient's condition. Those forms of chronic gastritis in particular that follow alcoholic excesses and excessive smoking seem to be benefited. The same probably applies to other salt waters, as Wiesbaden, for instance. The Glauber salt springs probably exercise the same effect as the alkaline and alkaline-muriatic waters; possibly the former are capable of producing a little more active evacuation than the latter.

From all this we learn that alkaline, alkaline-muriatic, and Glauber salt waters are indicated particularly in those forms of gastritis that are characterized by an increased or only slightly decreased production of hydrochloric acid. The salt waters of Kissingen, Homburg, and Wiesbaden, on the other hand, are useful particularly in those cases in which the secretion of gastric juice is reduced. In the majority of cases of chronic gastritis, therefore, salt waters are the most useful, and should be considered in the first place. The quantity of water to be given naturally varies in each case; if there are atony and dilatation, large quantities should be avoided. The temperature of the water is also important, as we have said above. Water that is too hot should be cooled, other waters should be slightly warmed. In general it is best to give the water lukewarm in all cases of chronic gastritis.

If necessary a course of waters may be given at the home of the patient, and in poor patients the mineral waters may be replaced by Sandow's salts.

Medicamentous Treatment.—The most important factor in the treatment of stomach cases is the administration of a diet that can be digested, and that is selected to fit the digestive powers of the stomach. The second factor in importance is lavage, either with plain water or with solutions of certain remedies that can dissolve mucus. The third factor is mineral waters, and of these the salt waters are the most important. The two last-named methods, although they may be of great advantage in many cases, can usually be dispensed with. A careful regulation of the diet should, however, always be insisted upon. In many cases these different measures alone will suffice.

In former days every case of chronic gastritis was treated with a large number of drugs, but nowadays the internal administration of medicine plays a subordinate role as compared to the other methods we have described. Drugs are usually given not to cure the disease, but to relieve certain symptoms.

Among those symptoms that may require special medicamentous treatment, loss of appetite is one of the most important. The appetite is frequently lost a short time after methodic lavage is instituted. In some cases the use of salt waters, particularly if the course of treatment is taken under favorable conditions and in a watering-place, has a tendency to improve the appetite. If this is not the case, and if anorexia persist, stomachics may be tried. One of the best is condurango; that may be given either in the form of the decoction, of the maceration, or of the wine of condurango. The dose of the latter should be a teaspoonful several times a day. The fluid extract of condurango, 25 to 30 drops several times a day, may also be given before meals. It is best not to mix the decoction of condurango with a syrup; a little hydrochloric acid added to the decoction is, however, beneficial. Orexin may also be tried, but only in those cases where the secretion of gastric juice is reduced, and not where there is hyperacidity. This remedy has slight irritating properties, and should be given carefully and only in the form of the basic preparation. The dose should be 0.2 gram, preferably given with meat broth.

Other remedies that are used to improve the appetite are creosote, tincture of nux vomica, the compound tincture of china, tinct. amara aromatica, etc. Strong alcoholics should be avoided, also strong spices. The majority of so-called stomach elixirs are to be condemned.

Hydrochloric acid may occasionally act as a stomachic. This drug, as we know, is used for three purposes in the treatment of stomach diseases: 1, to replace absent or deficient hydrochloric acid; 2, to disinfect the stomach-contents; 3, as a stomachic. I have already explained in detail, in the general part of this work, that hydrochloric acid administered by mouth rarely compensates the deficiency in cases where the secretion of hydrochloric acid is deficient. The same applies to its antizymotic action when it is given in small doses. As long as the motor power of the stomach is intact, there will be no fermentation nor decomposition, even though hydrochloric acid be absent; and, inversely, the ordinary small doses of hydrochloric acid fail to stop fermentation and decomposition if there is much stagnation of stomach-contents. The chief effect, therefore, of hydrochloric acid is stomachic. We do not know how this effect is exercised; experience, however, has taught us that hydrochloric acid is useful in this direction. I have found that 8 to 10 drops in a wineglassful of water before eating is the best method of administration.

Frequent vomiting is rare in chronic gastritis. Wherever this occurs methodic lavage of the stomach is the most rational treatment. Gastric pain is rarely a conspicuous symptom, and usually disappears if the diet is carefully regulated and the above methods of treatment are instituted. If the pain persists and is very severe, Priessnitz compresses or possibly poultices may be used. Narcotics are, as a rule, superfluous. If they are given at all, they should be employed in the form of suppositories.

Belching is occasionally a disagreeable symptom. It usually yields rapidly to methodic lavage. In other cases this treatment only relieves it partially. The administration of alkalis, bicarbonate of sodium or burnt magnesia, frequently helps. These drugs had best be given in capsule. If there is much gaseous fermentation at the same time, I am in the habit of adding a few grams of salicylate of sodium. Some physicians prefer to give these drugs in the form of tablets or lozenges.

More or less obstinate constipation is quite frequently seen in chronic gastritis. If this condition persists despite the administration of a suitable diet and the abundant ingestion of stewed fruit and of mineral waters, irrigation should be tried. Laxatives are contraindicated, for they only impede the cure of the primary disease, the gastritis. In many instances there is atony of the colon, a condition that can only be recognized by inspection. If this is the case, massage may be used. These two methods, irrigation and massage, will usually accomplish the desired result.

In conclusion, I must mention a few remedies that we employ more to replace deficiencies in the gastric juice than to stimulate its secretion. I refer to hydrochloric acid, pepsin, papain, papayotin, and the preparations of pancreas.

I have already stated that hydrochloric acid is frequently used as a stomachic, and that it may act favorably in this respect. Pepsin is only absent in very severe forms of gastritis, and not in the milder cases. If pepsin is absent, hydrochloric acid is also always absent. As it is impossible to supply a sufficient quantity of hydrochloric acid, it is also impossible to raise the digestive powers of the stomach by administering pepsin and hydrochloric acid together. Papayotin and preparations of papain have also been recommended. Sittmann[1] speaks favorably of papain (Reuss), and claims that it is an excellent remedy for restoring the disturbed chemical functions of the stomach. This author unfortunately fails to report detailed investigations on the effect of this remedy upon gastric secretion and absorption. Grote[2] has performed a number of experiments in my clinic that show that no particular gain is derived from the administration of papain.

Other authors recommend preparations of pancreas. These, of course, must always be given together with sodium bicarbonate. Theoretically, these remedies are indicated only in cases where the power of gastric digestion is completely lost. Here pancreatic digestion would, so to speak, be carried on in the stomach, and this presupposes that the acidity of the stomach-contents is completely neutralized by the alkali that is administered. These arguments alone show that pancreas preparations are hardly ever indicated in chronic gastritis, for here there is usually only a slight decrease in the secretion of gastric juice. We have, however, tried pancreas preparations in cases where the gastric secretion was altogether lost, but failed to see any particular benefit. This is readily explainable, for as long as the motility of the stomach is good apepsia does no harm, as the small intestine vicariously assumes the functions of the stomach. In cases of this kind, therefore, preparations of pancreas are unnecessary. If, on the other hand, there is much atony, then fermentation and decomposition of the stomach-contents occur, and here again pancreas given by mouth can do no good.

All these substitutes for gastric and intestinal products are without value. This includes the wines of pepsin that are lauded so much. As a matter of fact, large quantities of the latter[3] may directly weaken the power of the stomach to digest albumin.

ATROPHY OF THE GASTRIC MUCOSA; ACHYLIA GASTRICA.

(Atrophic Gastritis, Phthisis Ventriculi, Atrophy of the Stomach, Catarrhus Atrophicans, Anadeny of the Stomach.)

For the older literature I refer to Leube, in Ziemssen's *Hand-Book of Special Pathology*, 1878. In the following summary only the more important of the modern investigations are given.

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[1] *Münch. med. Wochenschr.*, 1893, No. 29.
[2] *Deutsch. med. Wochenschr.*, 1896, No. 30.
[3] Compare Werther, *Berlin. klin. Wochenschr.*, 1892, No. 27.

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Nature and Etiology.—Chronic gastritis may be cured, but may also lead to a complete loss of the glandular apparatus of the stomach, to atrophy of the mucosa. If the secreting parenchyma perishes, its product, gastric juice, is naturally absent. This absence of gastric secretion is called achylia gastrica. The term "anacidity" appears to me less suitable, for it indicates merely the absence of acid and not the absence of ferments. Inversely the stomach-contents may be very acid from the presence of organic acids even though the secretion of gastric juice is completely inhibited. Only if the gastric juice is anacid can we speak of the loss of hydrochloric acid production.

Fenwick,[1] in 1877, was the first to call attention to atrophy of the gastric mucosa. He deserves credit for having recognized this condition as an independent disease, and for having stimulated investigation into the relation between atrophy and essential pernicious anemia. A large number of investigators have busied themselves with a study of this disease. I merely mention Quincke,[2] Nothnagel,[3] Ewald,[4] Lewy,[5] Thorogwood,[6] Jaworski,[7] Boas,[8] Rosenheim,[9] Litten,[10] Einhorn,[11] Eisenlohr,[12] Schmidt,[13] and Martius.[14] It would lead us too far to discuss the investigations of all the authors that have contributed to our knowledge of this subject.

Atrophy of the gastric mucosa is by no means as rare as is ordinarily believed. This condition manifests itself clinically by a loss of gastric secretion that may be complete or only partial. In the latter instance we speak of achylia gastrica. Even those cases in which the secretion of gastric juice is more or less decreased are not, however, necessarily cases of atrophy of the gastric mucosa; some authors have diagnosed atrophy of the gastric mucosa and a complete cessation of gastric secretion whenever they found free hydrochloric acid absent. This is going too far, for we must consider not only the free hydrochloric acid, but also the combined hydrochloric acid. If the stomach-contents is neutral after a test-breakfast or a test-meal, or if there is only slight total acidity, for instance 4 or 6, this does not necessarily imply that the secretion of gastric juice or of hydrochloric acid is completely lost, and more careful examination is urgently called for. If there is anacidity, however, we can always diagnose achlorhydria. If we find acid stomach-contents, but no free hydrochloric acid, then the question must be decided by a careful quantitative determination of the chlorin. Only if chlorin is absent can we speak of a loss of hydrochloric acid secretion. Unfortunately, many of the cases reported in the literature are valueless, because the different investigators considered only free hydrochloric acid.

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[1] *The Lancet*, 1877; *Virchow's Arch.*, vol. cxviii.

[2] *Volkmann's Samml. klin. Vorträge*, No. 100.

[3] *Deutsch. Arch. f. klin. Med.*, 1879, vol. xxiv.

[4] *Berlin. klin. Wochenschr.*, 1886, No. 32; 1892, Nos. 26, 27.

[5] *Ibid.*, 1887, No. 4, and *Ziegler's Beiträge zur pathol. Anat. u. Physiol.*, 1886.

[6] *Med. Times and Gaz.*, 1881.

[7] *Münch. med. Wochenschr.*, 1887, Nos. 7, 8.

[8] *Ibid.*, Nos. 41, 42.

[9] *Berlin. klin. Wochenschr.*, 1888, No. 37.

[10] *Deutsch. med. Wochenschr*, 1888, No. 37; *Zeitschr. f. klin. Med.*, vol. xiv.

[11] *New York. med Monatsschr*, July, 1892; *New York Med. Record*, June, 1894

[12] *Deutsch. med. Wochenschr.*, 1892, No. 49.

[13] *Ibid.*, 1895, No. 19.

[14] Martius, *Achylia gastrica, ihre Ursachen und ihre Folgen mit einem anatomischen Beitrag von Prof. Lubarsch*, Leipsic and Vienna, 1897.

Achylia gastrica is seen as an apparently independent perversion of function following atrophy of the gastric mucosa or a number of other diseases, as carcinoma. It is also occasionally seen in the terminal stages of chronic gastritis. In carcinoma there is rarely complete achylia, but usually hypochylia—that is, there is more or less reduction in the secretion of gastric juice. Achylia and hypochylia gastrica have also been seen to follow diseases of remote organs.

The condition, however, is seen most frequently in carcinoma of the stomach. In this disease degenerative changes in the gastric mucosa usually develop in the later stages; they start from the immediate vicinity of the carcinoma and finally damage the glandular apparatus of the stomach over wide areas, so that there is more or less pronounced atrophy of the whole mucosa. This explains the gradual reduction in the secretion of gastric juice (hypochylia) that may ultimately lead to complete achylia.

While this may explain the severe degenerative changes in the secreting parenchyma of the stomach in advanced cases of carcinoma and the more or less complete loss of gastric secretion that results therefrom, it does not explain why small circumscribed carcinomata in their earlier stages frequently lead to a great reduction in the secretion of gastric juice. This peculiar phenomenon leads many investigators to speak of a remote or a toxic effect of carcinoma on the rennet glands. In favor of this view the argument has been brought forward that hypochylia and achylia gastrica with atrophy of the gastric mucosa are quite frequently seen in carcinoma of remote organs.

As early as 1880 Fenwick[1] called attention to the occurrence of atrophic processes in the gastric mucosa in carcinoma of remote organs, particularly of the breast, the intestine, and the uterus. Ewald,[2] in 1886, reported a case of carcinomatous ulcer of the duodenum in which he found atrophic degeneration of the gastric glands of the fundus and the cardiac portion of the stomach, and, besides, a connective-tissue proliferation of the pyloric portion of the organ with loss of glandular substance. Analysis of the stomach-contents during life showed that free hydrochloric acid was constantly absent, and that abundant quantities of lactic acid were always present. I[1] also called attention to the fact that in carcinoma of the esophagus the peptic powers of the stomach are occasionally very much reduced. Many authors claim that this hypochylia or achylia is due to the serious disturbance in the general nutrition of the patient that is caused by carcinoma, for in many diseases that are complicated by general cachexia atrophy of the gastric mucosa ultimately develops. I do not consider this view correct, particularly in small circumscribed carcinomata, for here the general condition of the patient may be excellent and still the peptic powers of the stomach be very much reduced in the earliest stages of the disease. Those cases of general senile marasmus may possibly be explained on the basis of general nutritional disturbance in which atrophy of the gastric mucosa is seen, but even in those cases there is rarely total atrophy, but usually merely partial atrophy.

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[1] *On Atrophy of the Stomach*, London, 1880.

[2] *Berlin. klin. Wochenschr.*, 1886, No. 32.

Rosenstein[2] found atrophy of the gastric mucosa in a few cases of diabetes mellitus, but this finding is by no means constant. Honigmann[3] examined 8 cases of diabetes in my clinic, and found the stomach perfectly normal in one case, hyperacidity in three cases, absence of hydrochloric acid in three cases, and fluctuating findings in one. In cirrhosis of the liver secondary atrophy of the gastric mucosa has repeatedly been observed.

Atrophy of the mucous membrane of the stomach is quite frequently seen as an independent disease. It occurs with relative frequency as the result of direct damage to the stomach by some toxic substance; in other words, as the result of toxic gastritis. As early as 1886 I[4] reported a case in which sulphuric-acid poisoning led to pronounced atrophy of the mucous lining of the stomach with formation of cicatrices, and in which the peptic powers of the stomach were almost completely lost during the life of the patient. Chronic parenchymatous gastritis also occasionally terminates in atrophy. The interstitial form that primarily leads to hypertrophic proliferation of interstitial tissues may also ultimately lead to complete atrophy of the mucous membrane. In cases of this character the stomach may ultimately become cirrhotic and reduced in size. This was shown by Nothnagel[5] in a well-known case that he reported.

In cases of severe pernicious anemia postmortem examination of the stomach frequently reveals so-called loss of the rennet glands. Fenwick was the first to report such an interesting case, and since his day a number of analogous observations have been reported. Until quite recently there was a great diversity of opinion in regard to the relation between this glandular degeneration and the serious blood-changes that occur—that is, it remained undecided which was cause and which was effect. The majority of authors were inclined to consider the glandular degeneration as the primary factor. It is undoubtedly true that total atrophy of the gastric glands may lead to serious disturbances of the general health; on the other hand, it has been demonstrated that a complete loss of peptic power may be tolerated for many years without particular impairment of the general health provided that the motor power of the stomach remains intact and the intestine can vicariously assume the functions of the stomach. As soon, however, as atony is associated with loss of peptic power nutrition must suffer. [Considering the embarrassment to the digestion produced by achylia gastrica, it is easy to understand why the atrophy of the gastric mucosa found in advanced cases of progressive pernicious anemia was held to be responsible for the blood-changes. Nevertheless, such a conclusion is erroneous. The severe anemias that sometimes are present in achylia gastrica are found upon careful study of the blood to be secondary anemias. In a large group of cases of pernicious anemia in which the gastric contents had been carefully examined, there were a number in which, in the early stages of the anemia, the gastric secretion was not affected, but as the intensity of the anemia increased, the secretion of the stomach decreased, and when the anemia reached a severe grade the gastric secretion entirely disappeared. It seems improper to consider achylia gastrica the cause of pernicious anemia, and if the achylia gastrica precedes a primary anemia, it is to be regarded as coincident. —Ed.] In anadeny other dangers threaten, for fatty degeneration of the mucosa of the intestine and fatty degeneration of the muscularis frequently develop with atony of the gastric mucosa. Even if a simple acute intestinal catarrh is superadded to gastric atrophy, the condition of the patient becomes precarious, for as soon as the intestinal functions are disturbed nutrition naturally suffers.

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[1] *Zeitschr. f. klin. Med.*, vol. xi.

[2] *Berlin. klin. Wochenschr.*, 1890, No. 13.

[3] *Deutsch. med. Wochenschr.*, 1890, No. 43.

[4] *Zeitschr. f. klin. Med.*, vol. xi.

[5] *Deutsch. Arch. f. klin. Med.*, vol. xxiv.

We see, therefore, that, notwithstanding complete atrophy of the gastric mucosa and complete loss of the peptic power of the stomach, nutrition may remain undisturbed provided the motor power of the stomach remain intact and the intestine performs its normal functions. Von Noorden[1] showed in a number of cases that he examined in my clinic and in several other cases from Gerhardt's clinic that even though the hydrochloric acid secretion of the stomach is greatly reduced—that is, if conditions obtain that must lead to an almost complete inhibition of gastric digestion—the assimilation and absorption of the food, particularly of the proteids, can take place to a fair degree. This fact has been verified by repeated clinical observation. Einhorn[2] reports a case of achylia gastrica (as he is in the habit of calling that condition in which the stomach does not produce any gastric juice) that he observed for four years, and whose general health if anything improved. He also reported a second case the history of which seemed to indicate that the patient's stomach was in this condition for some forty years. In the latter case no symptoms whatever were complained of, and both patients seemed capable of eating the most indigestible food with impunity.

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[1] *Zeitschr. f. klin. Med.*, vol. xvii.

[2] *Medical Record*, June, 1892.

I have observed a number of these cases myself, among them one in which I could demonstrate twelve years ago that the peptic powers were lost, and in which complete inhibition of peptic digestion persists to this day. Ewald[1] also reported a case in which the secretion of gastric juice was stopped for two years and a half; nevertheless, this patient gained 42 pounds. We are never justified, as I have said above, in concluding that there is atrophy of the gastric mucosa if the peptic powers of the gastric juice are lost. If there is atony, there must, of course, be achylia gastrica—that is, the secretion of gastric juice must cease. It is an open question, however, whether or not this inhibition of gastric secretion can occur in some other way, for instance, after certain nervous disturbances. Martius in his most recent work distinguishes two forms of achylia gastrica: one that is due to atrophy of the mucosa, another that he considers a primary defect in the secretory powers of the organ. He argues that the latter form is either congenital or at least develops on the basis of some congenital predisposition. It is found chiefly in neurasthenics. He also states that the gastric mucosa in these cases possesses less resisting power against external agencies than the normal gastric mucosa the secretion of which is not reduced. This explains why anatomic changes of mild and severe degree that are in no proportion whatever to the absolute loss of function are nearly always found in the latter form of achylia gastrica simplex. Small pieces of mucosa removed with the sound in these cases always showed more or less granular degeneration. It is not permissible, of course, to apply the finding in isolated pieces of mucosa to the whole mucous lining of the stomach. For the present we have no autopsy reports on cases that died during the earlier stages of this disease, and in which the changes in the whole mucosa were probably slight, even though the secretion of gastric juice may have been completely or nearly completely stopped during life. All these observations, at all events, teach us that we are not justified in diagnosing total atrophy of the gastric mucosa even in those cases where the clinical picture of achylia gastrica is presented. It cannot be denied that occasionally achylia gastrica may be merely a perversion of function. [The belief that achylia gastrica may in some instances begin as a simple depression of the functional activity of the secretory glands of the stomach, the result in some cases of neurasthenia, in others of reflex irritation, appears to be gaining rather than losing ground. For a time, with improvement in the general health, the gastric acid is raised, but subsequently declines if the nerve strain is reëstablished. When the secretion has been for a long time absent, it may be assumed that much atrophy of the mucosa has resulted from lack of use, and yet, in exceptional cases, as I have myself determined, the secretion returns after it has been absent for a period of a year or more.—ED.]

Achylia gastrica is most common in the middle and later years of life. Occasionally it is found also in younger people. Nothnagel's[2] patient was twenty-three years old. Litten and Rosengart[1] report a case of this disease in a girl of eighteen years. One of Einhorn's[2] patients was twenty-five years old. Von Noorden's[3] case from my clinic was a girl of nineteen years. We have recently reported a case in a strong youth of seventeen years. So far no difference in the frequency of this disease in the two sexes has been determined.

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[1] *Berlin. klin. Wochenschr.*, 1892, Nos. 26, 27.
[2] *Deutsch. Arch. f. klin. Med.*, 1879, vol. xxiv.

[On the question of the nature of achylia gastrica E. Kuttner[4] states that at present he considers it impossible to differentiate the clinical picture of the simple achylia gastrica from that of absence of secretion due to advanced catarrhal or atrophic changes of the gastric mucosa. The individual symptoms are not only not pathognomonic, but a functional achylia may pass into an organic disease. We have no method of determining whether a reduction in secretion is due to changes in the mucous membrane or to purely nervous influences. Kuttner has no faith in the results of examinations of fragments of mucous membrane removed by lavage, and says that as his experience increases he attaches less value to the findings. He agrees with Leuk, who held that in normal digestion one may find particles of mucous membrane that show marked degenerative changes, while in a large percentage of cases in which there are very decided anomalies of secretion one may find fragments of the mucosa that are apparently normal.

Einhorn[5] has published a review of this subject, including some recent work of his own, and concludes as follows: "The secretory functional disturbances of the stomach are not based on a primary change in the mucous membrane of the stomach. They rather produce, if they last for a long time, lesions of the mucosa of greater or less extent."—Ed.]

Anatomic Changes.—Numerous differences are found in the anatomic lesions observed. In some cases the stomach is dilated, in others it is of normal size, and in still others it is smaller than normal. In several instances there was gastroptosis at the same time.

The wall of the stomach may be of normal thickness, or it may be abnormally thin or thick. In the majority of cases the wall of the stomach was very much thinner than normal, particularly where there was ectasy at the same time. In some instances it was as thin as a piece of paper. Wherever it was found thickened there were proliferation of the glandular connective tissue and hypertrophy of the muscularis and of the submucosa. Atrophy of the gastric mucosa, in which there is complete loss of secreting glandular epithelium, usually represents the terminal stage of some inflammatory affection of the organ. In ordinary chronic gastritis the infiltration of the interglandular connective tissue and the cloudy swelling of the glandular cells, with fatty degeneration and complete loss of glandular parenchyma, may lead to a complete loss of the whole glandular layer of the stomach, so that nothing remains but a narrow layer of small round cells that here and there enclose remnants of parenchymatous cells. In other cases, again, the inflammation involves the connective tissues that surround the gland tubules and leads to the development of hyperplasia of the interglandular tissues. This condition we have already spoken of in discussing gastritis. In the earlier stages remnants of glandular tubules are seen between the trabeculæ of connective tissue; but finally the latter disappear entirely. Occasionally, when the inflammatory process follows the latter course, the muscularis is also involved; whereas when it follows the former course the muscularis usually remains intact. Both forms may merge into one another. Toxic gastritis may also occasionally terminate in atrophy.

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[1] *Zeitschr. f. klin. Med.*, vol. xiv.
[2] *Arch. f. Verdauungskrankh.*, vol. i., No 2.
[3] *Zeitschr. f. klin. Med.*, vol. xvii.
[4] *Ibid.*, vol. xlv., Nos. 1, 2.
[5] *Amer. Jour. Med. Sciences*, October, 1902.

On microscopic examination the mucosa will be found grayish-yellow in color, thin, with a smooth surface that resembles the serous membrane. The folds of mucous membrane will be barely outlined, and the whole mucosa will be detached from its base so that it is freely movable. This condition is, of course, found only in pronounced cases. In other instances a certain amount of glassy mucus will be found on the surface of the mucosa; in other cases, again, this mucus is absent.

Microscopic examination shows that the glandular elements are almost completely or completely lost, and this finding is characteristic. Here and there a few isolated glands that have not yet undergone cystic degeneration may be found. The connective tissue of the atrophic mucosa is always more or less changed, usually hyperplastic. The ordinary gastric epithelia have changed their form. Cylindric epithelia, that frequently assume the shape of goblet cells, are seen both in the different channels of the glands and in the deeper layers of the mucosa.

In some instances the lesions are seen only in the mucosa, whereas the other layers of the stomach and, particularly the muscularis, remain intact. This was the case, for instance, in the stomach that Schmidt[1] examined. When the muscularis is intact, there is no disturbance of the motor powers of the stomach, and the gastric contents are propelled into the intestine within the normal time-limits.

In other cases the sclerotic changes extend to the submucosa, the muscularis, and under certain circumstances to the serosa. Whereas we are justified in the first class of cases in assuming that atrophy of the glands is the primary feature of the disease, we cannot tell in the second group whether or not the glandular atrophy is primary or secondary, with presumptive evidence in favor of the latter supposition. The two forms probably merge into one another. At the same time there is no doubt that we occasionally, though rarely, encounter an interstitial form of inflammation of the stomach in which the sclerotic process starts from the peritoneum. In the majority of cases of atrophy, however, we must assume that the primary cause of the disease affects the surface of the mucosa and its surface epithelium in the first place, and that from here the process extends downward into the other tissues of the stomach-wall.

Most authors agree in considering those diseases that may lead to cachexia as the most prolific cause of the secondary forms of atrophy of the gastric mucosa. It is frequently impossible, however, to find the direct cause of the primary forms of this affection. Some investigators believe that trophic disturbances of the gastric mucosa that may be due to some nervous disorder can lead to atrophy. Jürgens,[1] Blaschko,[2] and Sasaki[3] have reported a number of findings that speak in favor of this supposition. They found fatty degeneration of Meissner's and Auerbach's plexuses in a number of cases of gastro-intestinal atrophy. Meyer[4] emphasizes the fact that possibly isolated atrophy of the stomach may occasionally be caused by anatomic changes in the nerves of the stomach, just as atrophy of the gastro-intestinal mucosa is found in diseases of the plexus of Meissner and that of Auerbach.

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[1] *Deutsch. med. Wochenschr.*, 1895, No. 19.

In a few cases of gastric atrophy interesting lesions of the spinal cord have been seen; it is undecided what relation they bear to atrophy—that is, whether they cause atrophy, or whether, as Eisenlohr believes, they are the result of secondary anemia.

Symptoms.—The symptoms of atrophy of the gastric mucosa, or more correctly of achylia gastrica, are in no way characteristic. As a matter of fact, there are cases of this disease in which no gastric symptoms develop, and in which the appetite remains unimpaired. At the present time, for instance, I have two cases of achylia gastrica in my wards that complain of no stomach symptoms whatever. Other cases that are complicated with this disease occasionally develop a very serious train of symptoms. In many instances the appetite is greatly reduced, and the patients, like cancer cases, have a marked aversion to meat. Pain is usually absent; in other instances the patients complain of a feeling of pressure, heaviness, and fulness in the stomach, and in still others there are occasional paroxysms of violent pain. These attacks usually occur after eating, generally very soon after a meal; in general they persist for some time, occasionally for several hours.

In some cases the pain becomes very intense, more intense than in simple chronic gastritis. Even this can hardly be considered a pathognomonic symptom, for attacks of pain are not constantly found in atrophy, and may occur in many other affections of the stomach.

Vomiting occasionally occurs, but is not a constant symptom. In some instances it occurs very soon after eating, particularly in cirrhosis of the stomach proper with reduction in the size of the organ; in other cases it occurs some time after eating. The vomit almost always consists of coarse undigested remnants of food. Vomiting of blood has never been observed in atrophy of the stomach. Belching, on the other hand, is a common symptom.

Many patients complain of headache and vertigo. The stools are usually sluggish. Diarrhea is rare; occasionally diarrhea alternates with constipation. A few cases are reported in which the attacks of diarrhea were the most important symptom that the patient complained of. Oppler[1] has recently reported a number of these cases in which all the symptoms pointed to some affection of the intestine, and not of the stomach, and in which the impression was created that the patient was afflicted with some very serious intestinal disease. On careful examination of the stomach and its contents it was found, however, that all peptic power was lost. Patients of this kind suffer from frequent attacks of diarrhea, of a feeling of discomfort and tension in the abdomen, of gurgling in the intestines, but do not complain of any gastric symptoms. The appearance of these cases varies greatly. [The frequency with which cases of achylia gastrica are attended with lienteric diarrhea is a question which has been considerably discussed in this country, and on which observers differ. In my experience diarrhea is more common than constipation, and in a number of instances, as pointed out by Dr. Allen Jones,[2] the nature of the case was rightly predicted merely from the character and the persistence of the diarrhea occurring soon after meals, especially after breakfast.

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[1] *Berlin. klin. Wochenschr.*, 1882, No. 28.

[2] *Virchow's Arch.*, vol. xciv.

[3] *Ibid.*, vol. xcvi.

[4] *Zeitschr. f. klin. Med.*, vol. xvi.

Max Einhorn[3] has called attention to the fact, which must have been recognized by all having much experience in stomach diseases, that there are occasionally cases of achylia gastrica that present symptoms that are quite parallel to those of hyperchlorhydria.—Ed.]

Subjects may be afflicted with this disease for many years and still appear well nourished. This, as we have said, is only possible if the muscularis of the stomach is not involved, for only then can the ingesta be propelled into the intestine within a short time, and only if this occurs can the intestine vicariously assume the digestive function of the stomach. In other cases, again, the general health of the patient is seriously impaired, and there is perversion of gastric function. Some patients develop neurasthenic symptoms of all kinds in achylia gastrica, and occasionally these are so prominent that they dominate the disease-picture.

Those cases in which atrophy of the gastric mucosa presents the syndrome of progressive pernicious anemia are particularly interesting. The patients present the symptom-complex of severe anemia ; the skin and the visible mucous membranes are extremely pale, the number of red blood-corpuscles is considerably reduced, the hemoglobin is also much reduced, and there is poikilocytosis, etc.

This picture of severe anemia is not, however, seen only in those cases in which the assimilation of food is impaired as a result of atony of the gastric mucosa ; it is seen also in cases in which the tone of the stomach is fairly well preserved. In the latter cases all symptoms that indicate serious disease of the stomach may be absent, so that the true nature of the disease remains unrecognized until the stomach-contents is aspirated and carefully analyzed. I had occasion to study a case of this kind quite recently. The patient was a woman of thirty-three years, of strong build, and apparently suffering from severe anemia. The skin and mucous membranes were very pale, the pulse was small and thready; at the same time the adipose layer was well developed. Gastric symptoms like cardialgia, vomiting, etc., were absent. The appetite was irregular. The stomach-contents was aspirated on different occasions and analyzed. It was found that all peptic power was lost, that both hydrochloric acid and pepsin were absent. The total acidity was slight throughout, fluctuating between 2 and 6. The motor power of the stomach, however, was intact, for the stomach was always found empty within four hours after a test-meal. When the patient entered the hospital there was slight diarrhea, but this was soon checked. After a short time the appetite improved, the patient tolerated all kinds of food, and her general appearance improved, but notwithstanding all this the peptic power of the stomach remained almost completely lost and the acidity of the gastric juice fluctuated between 3 and 6. There is no doubt that in this case the attacks of diarrhea contributed much to the development of the anemia, for since the intestinal function was also disturbed, the assimilation of food and the formation of blood naturally suffered.

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[1] *Therapeut. Monatsh.*, 1896 ; *Deutsch. med. Wochenschr.*, 1896, No. 32.

[2] *Med. News*, December 15, 1894, and *Amer. Jour. Med. Sci.*, July 30, 1898.

[3] *Jacobi Festschrift*, 1901.

Ewald[1] noted that the symptom-complex of achylia gastrica is occasionally seen to develop in the course of acute febrile diseases, for instance, of influenza; in other words, that this condition of the stomach may apparently develop suddenly. I am inclined to believe that in these cases the achylia had existed for a long time, but that as soon as atony of the stomach or a catarrh of the small intestine developed the compensatory processes that had vicariously assumed the stomach functions suddenly became insufficient.

Even though the secretion of gastric juice is completely stopped for a long time, we can never diagnose the existence of anatomic lesions or of atrophy of the glands. Einhorn[2] in particular has shown that we frequently encounter cases in which the peptic powers of the stomach are permanently lost, but in which we are nevertheless not entitled to assume that the mucous lining of the stomach is altered.

Einhorn has carried out microscopic examinations of shreds of gastric mucosa in cases of this kind, and has been able to demonstrate that normal glands may be present even though there is complete achylia gastrica. Again, he saw a case of achylia gastrica that persisted without change for five years, and in which the condition improved and the secretion of gastric juice was resumed. In the latter case he succeeded in demonstrating that the acidity gradually increased, that the stomach-contents became more and more finely divided, and that finally free hydrochloric acid again appeared in the stomach-contents. Einhorn expresses the belief that this peculiar development can only be explained by assuming that the suppression of gastric secretion was not due to a total loss of the glandular layer, but to some nervous disorder. At all events, this case proves that the clinical picture of achylia gastrica does not demonstrate by any means that the glandular layer of the stomach is completely destroyed, and does not justify us in diagnosing anadenia ventriculi.

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[1] *Klinik d. Verdauungskrankh.*, third edition, p. 215.

[2] *Arch. f. Verdauungskrankh.*, vol. i., No. 2.

Martius, as we have mentioned, guided by his clinical observations, also arrived at the conclusion that in many of these cases there is no complete atrophy of the gastric mucosa, but merely a certain secretory weakness that is either congenital or develops from some congenital predisposition.

The following objective changes are seen : In many cases the stomach is enlarged to a considerable extent, in others it is only slightly enlarged. Advanced degrees of ectasy are rarely observed in this condition ; the latter are principally found in the secondary forms of atrophy, particularly in those that are due to carcinomatous strictures of the pylorus. Here, however, ectasy is not so much the result of atrophy as of the carcinomatous stenosis of the pylorus. Occasionally toxic gastritis is followed also by cicatricial stenosis of the pylorus with secondary ectasy.

The only way in which to arrive at a positive diagnosis is to analyze the stomach-contents. If repeated analysis of the chemism of the stomach is indicated anywhere, it is certainly indicated here, for only if the results of gastric analysis are uniform can we diagnose achylia gastrica or atrophy of the gastric mucosa. If the stomach-contents is aspirated an hour after a test-breakfast, only a small quantity of residue will, as a rule, be found. The morsels of bread that are removed are merely a little swelled. Free hydrochloric acid is completely absent, and the total acidity is also very slight. The gastric contents are usually slightly acid in reaction. The total acidity is usually 1 to 4, rarely more ; that is, 100 c.c. of the filtrate of gastric contents are saturated by from 1 to 4 c.c. of a $\frac{1}{10}$ normal sodium hydrate solution. This proves that there is no free hydrochloric acid in the stomach-contents, and that there is a great deficiency of the combined hydrochloric acid. It is unnecessary to perform quantitative estimations of combined hydrochloric acid in these cases unless the total acidity is higher than that given above ; in the latter instance an exact quantitative determination of the hydrochloric acid, or better of the total chlorin, is necessary ; also a determination of the organic acids. Lactic acid is, as a rule, absent, or present only in traces ; it is found in large quantities only where there is much ectasy.

Gaseous fermentation is seen in exceptional cases, and can, of course, occur only if the stomach is anacid. In order that this condition of the gastric contents be brought about there must be much motor insufficiency. I have a case of this kind under observation at the present time. Here there is atrophy of the gastric mucosa that was caused by an intoxication, and at the same time there are ectasy and gaseous fermentation.

The reactions for peptone and propeptone usually give negative or very minimal results in pure cases of achylia gastrica. The gastric juice, even though a sufficient quantity of hydrochloric acid be added—that is, until the reaction for free hydrochloric acid appears—is incapable of digesting a disc of albumin. Pepsin, therefore, is absent, as well as hydrochloric acid.

In simple cases of subacidity—that is, in cases in which the production of gastric juice is merely reduced—free hydrochloric acid is absent, but the secretion of pepsin is approximately sufficient; in anaciditas hydrochlorica, however, the formation of ferments is disturbed in the same way as the production of hydrochloric acid. In these cases, therefore, the tests for rennet-zymogen and rennet-ferment give negative results.

This absence of all the digestive secretions—of hydrochloric acid, of pepsin, and of rennet-ferment—is of great diagnostic importance. The best methods for determining pepsin quantitatively are the one proposed by Hammerschlag[1] and the method of Oppler.[2] The latter is very complicated.

Similar results are seen after a test-meal as after a test-breakfast. If the stomach-contents, in pure, uncomplicated cases, is removed within four or five hours, the stomach will be found empty; this indicates that the motor powers of the organ are intact. The latter symptom is unquestionably of great interest, for it shows that the ingesta are moved into the intestine within a normal time, even though they may be incompletely converted in the stomach.

If the stomach is pumped out early in the morning, before breakfast, nothing will be found. It is interesting to note that in cases of this kind traces of blood or shreds of mucous membrane appear in the wash-water. This seems to indicate that the gastric mucosa is particularly vulnerable.

In regard to mucus, most authors seem to emphasize that in atrophy of the gastric mucosa the secretion of mucus is impaired in the same way as the secretion of acid and enzymes. It is considered characteristic that mucus is absent from the stomach-contents, and is not found in the stomach after fasting. To judge from my personal observations, this does not apply to all cases; in the more advanced forms, where the gastric epithelia are completely destroyed, the secretion of mucus, of course, stops; in the earlier stages of the disease this is different. Schmidt[3] has shown that in this process the gastric epithelium is converted into a new form of epithelium that greatly resembles intestinal epithelium, and is never normally found in the stomach. According to Schmidt, this pathologic epithelium covers the atrophic parts of the surface of the mucosa, and fills the spaces left by the disintegration of the glandular cells. In this new form of pathologic epithelium the formation of mucus is different from that of ordinary stomach epithelium, but a very considerable quantity may be secreted. This epithelium is very resistant, but may also finally perish if the degenerative processes continue.

Schmidt, therefore, considers it dangerous to attribute the same significance to a cessation of the secretion of mucus as to the cessation of the secretion of hydrochloric acid, pepsin, and rennet. In rendering a diagnosis of atrophy of the gastric mucosa the continued secretion of large quantities of tough mucus, in cases where hydrochloric acid and pepsin are completely absent, always justifies the suspicion that the case is one of atrophy. In looking over my clinical notes I find that both forms can occur, cases of atrophy with the formation of mucus and cases of atrophy without; the presence of mucus, however, should never be considered a proof of the absence of atrophy.

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[1] *Internat. klin. Rundschau*, 1894, No. 39.

[2] *Centralbl. f. innere Med.*, 1896, No. 1.

[3] *Deutsch. Arch. f. klin. Med.*, vol lvii.

Einhorn calls attention to another point, namely, that only a small quantity of fluid is found in the stomach in cases of this kind one hour after a test-breakfast. He explains this phenomenon by assuming that no gastric juice is secreted and added to the water or tea taken with the test-breakfast; and that at the same time the chyle is more fluid and passes the pylorus more rapidly than usual.

The motor function of the stomach, as we have said, is never depressed in cases where atrophy is limited to the mucous membrane—that is, in pure, uncomplicated cases. If the muscularis is involved in the atrophic process, atony and ectasy of severe degree with all their well-known consequences may develop.

Sticker and Hübner,[1] and also Einhorn,[2] have established another remarkable fact, namely, that in the above cases, and also in cases of primary atrophy, the acidity of the urine is not reduced at the height of digestion as in normal cases, but remains more or less uniform.

The duration of the disease varies greatly. Some cases lead to a fatal issue within a few months; other cases live for many years in comparatively good health.

Prognosis.—The prognosis varies in individual cases. If atrophy is secondary, the prognosis, of course, depends on the primary disease. The cure of this condition is always impossible. Secondary atrophy in carcinoma has the same prognosis as carcinoma itself. If the carcinoma is inoperable, the prognosis is bad; if a complete resection is possible, a relative cure may be brought about—that is, the patient may continue to enjoy comparative well-being and may appear healthy even though he is afflicted with atrophy of the gastric mucosa. As long as the motor powers of the stomach remain good, and as long as the intestine continues to perform its functions in a normal manner, this defect does not necessarily cause any symptoms nor damage the patient.

Atrophy that is accompanied by the syndrome of progressive pernicious anemia always offers a prognosis that is at least doubtful. The prognosis of the genuine primary form of achylia gastrica is more favorable; this condition may persist for many years without serious impairment of the general health, provided, of course, the motility of the stomach remains intact and the functions of the intestine remain normal. Injury in the one or the other direction is immediately followed by serious disturbances of nutrition. Einhorn's observations seem to show that certain forms of achylia gastrica may be improved.

Diagnosis.—The diagnosis of achylia gastrica *per se* is easy. It is based on the analysis of the stomach-contents, the results of which we have described. The most important feature is the complete or the almost complete absence of hydrochloric acid and of gastric ferments. It is much more difficult to render a decision in regard to the significance of this achylia—that is, whether it is due to atrophy of the mucosa or whether it is a primary nervous secretory perversion of the stomach. Even if the latter form can be excluded, even if it can be positively determined that the absence of the digestive ferments is due to atrophy of the gastric mucosa, the question still remains to be decided, whether this atrophy is independent and primary, or whether it is secondary.

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[1] *Zeitschr. f. klin. Med.*, vol. xii.

[2] *Arch. f. Verdauungskrankh.*, vol. i., No. 2.

It has not yet been established beyond cavil whether or not there is such a thing as a nervous or a congenital form of achylia gastrica. If in any given case certain nervous symptoms appear, if the gastric symptoms develop in immediate connection with some nervous excitement, the disease may possibly be considered of nervous origin. This, however, is never conclusive, for nervous and neurasthenic symptoms may be present together with atrophy of the gastric mucosa, and at the same time not be causally related to it. If it could be determined in any one case that the perversion of gastric secretion fluctuates in intensity, this might constitute an important factor in the diagnosis of a nervous trouble. The absence of all local symptoms and of all gastric disturbances is of no value from the point of view of the differential diagnosis, for all subjective and local symptoms may be absent, and frequently are absent in subacidity and anacidity due to atrophy. A history of catarrh or of some other lesion, particularly of toxic conditions that may lead to anadeny and atrophy, is more important in arriving at a decision.

If nervous or congenital anacidity can be excluded, it remains to determine whether atrophy is an independent condition or whether it is secondary to some other disease, particularly carcinoma. This differential diagnosis may occasionally be very difficult. If the typical symptoms of carcinoma, the classical picture of cancer, are presented, the diagnosis is, of course, easy. Such symptoms are, a tumor in the region of the pylorus, ectasy of the stomach, cachexia, rapid emaciation, and vomiting of coffee-ground material. This syndrome is found in the terminal stages of cancer. We occasionally encounter the statement that a well-developed adipose layer always speaks against carcinoma and in favor of anadeny. This, however, applies only to carcinomata of long standing. In the beginning many patients with carcinoma have a good *panniculus adiposus* and show no evidence of cachexia. Vomiting of blood is a more important symptom, for it has never been observed in anadeny; at the same it must also be remembered that it is frequently absent in carcinoma, particularly in the early stages. As it is very important to render a diagnosis as early as possible—that is, at a time when the patients have not yet developed cachexia, when their nutrition has not suffered, when there is no lactic acid fermentation, and when the only direct symptoms are subacidity and anacidity—this feature is of little value. If subacidity and anacidity are found, we usually think of atrophy of the mucosa, but are unable to say whether this condition is primary or secondary—that is, whether or not it is due to the presence of a carcinoma that cannot be detected. I believe that in these difficult cases the most valuable criterion is furnished by the course that the disease pursues. Primary atrophy, as a rule, develops slowly and gradually, for it represents the terminal stage of a long-lasting chronic gastritis, or is the result of toxic gastritis. If the history of the case shows that the symptoms are of recent date, if the patient is of an advanced age, if no cause for achylia can be discovered, we are justified at least in suspecting carcinoma even though all other signs of this condition are absent.

In many instances, however, a careful study of all these points fails to give sufficient information to enable us to render a positive diagnosis, and it is frequently impossible to determine whether we are dealing with simple achylia gastrica or whether the functional perversion is due to pronounced atrophy of the mucosa.

The differential diagnosis between the condition under discussion and amyloid degeneration is not so difficult. It is usually an easy matter to exclude amyloid degeneration of the mucous lining of the stomach. It is true that in this condition, particularly if it is somewhat advanced, the secretion of gastric juice may cease. This was conclusively shown for the first time by a number of investigations that were carried on in my clinic. The facts, however, that amyloid degeneration occurs only as the result of certain chronic diseases, that it is never limited to one single organ, and that, as a rule, there is amyloid degeneration of the liver, the spleen, and the kidneys, render the diagnosis easy.

Treatment.—It might be assumed *a priori* that the chief indication for treatment would be to restore or replace the deficient or absent peptic power of the stomach. The matter, however, as we have seen above, is not so simple. We frequently encounter cases in which achylia gastrica persists for many years, and in which nevertheless digestion proceeds in a normal manner and the general nutrition does not suffer. These are the cases in which the motor power of the stomach remains intact, in which there is no stenosis of the pylorus, and in which all the gastric contents are poured into the intestine as though they had been digested in the stomach in a perfectly normal manner. Here motility is, so to say, increased, and this process aids in compensating the loss of peptic power, so that all the disadvantages of achylia gastrica are compensated by the increased motor power of the diseased organ. If the motor power becomes impaired, the disease grows more serious, for the general nutrition suffers chiefly because the lack of peptic power is no longer compensated.

Acting on this well-recognized fact, our first endeavor in treating these cases should be to maintain the power of the stomach at as high a degree of efficiency as possible, and to raise it in all those cases in which it threatens to become impaired or is already impaired. This is the only means at our disposal to compensate the loss of peptic power.

We have already mentioned a number of very instructive cases that demonstrate the validity of this argument very forcibly, namely, those cases in which diarrhea occurs. Frequently patients of this character consult a physician for diarrhea. If the stomach-contents is carefully examined, it will be found that there is complete apepsia. As long as the intestine was intact and could perform its function in a normal manner no symptoms were produced; as soon, however, as the intestine became irritated by some unforeseen accident, possibly from being overtaxed, and consequently became insufficient, certain symptoms appeared that remained chiefly intestinal and not gastric. Nevertheless all these cases should be treated for stomach trouble. Here as in any other form of gastric anacidity we should endeavor above all to select a rational diet, chiefly a diet that furnishes enough nutriment. The most important factor is the form in which the food is administered.

The food should be as finely divided as possible, and had best be given in the form of mushes and liquids. If there are symptoms of irritation in the intestine, if there is diarrhea, a diet of soup should be given for several days. Such soups may be made of barley, oatmeal, rice, or sago; vegetables usually agree very well. Cereals, of course, should be given as flour after removal of all cellulose. Leguminous plants are very useful owing to the large proportion of albumin that they contain. Mushes made from rice, tapioca, groats, potato, peas, lentils, oats, aleuronat, and white flour, leguminose, etc., are all to be recommended. To these preparations somatose, nutrose, and similar preparations may be added. Puddings made from these flours are also good. Eggs may be given with advantage; they should be soft boiled and warm, or may be given as yolk of egg added to soup.

Fat in the form of butter is very good, provided there are no symptoms of intestinal irritation. Milk is not borne well by all cases, and should be given only in small quantities. Cream is also useful, and should be given as long as there are no symptoms of atony nor of stagnation of stomach-contents. There is no objection to coffee and tea with milk. But "Hafer" cocoa and "Kraft" chocolate are better.

As meat is not digested in the stomach, it might be argued that it had better not be given in these cases. As long, however, as the motor powers are intact, there is no good reason why it should not be given; it should, however, always be administered in small portions, and in a form that enables the stomach to propel it rapidly into the intestine,—that is, it should be finely chopped or scraped. The best meats are chicken, pigeon, finely scraped raw ham, young veal, calves' brain, roast beef, tender beefsteak, game, fish, etc.

Alcohol should be administered only if there is some direct indication for its exhibition, and then only in small quantities, preferably in the form of wine. Beer is not to be recommended.

The meals should not be very abundant, but should be given at frequent intervals. The patient should eat something about every three hours. It is, of course, very important to see that the patient receives a sufficient quantity of nourishment, a quantity that contains an adequate number of calories.

See diet-list on page 541, quoted from Wegele (*Dietetic Treatment of Gastro-intestinal Diseases*) that is suitable for cases of atrophic catarrh:

	Albumin.	Fat.	Carbohydrate.	Alcohol.
In the morning:				
150 gm. of maltoleguminose cocoa	6.0	4.0	13.5	
In the forenoon:				
100 gm. of wine	. .	. .	4.0	12.0
10 gm. of butter on toast	0.15	16.6	0.12	
At noon:				
100 gm. of maltoleguminose soup	2.6	0.1	6.2	
100 gm. of scraped beefsteak	20.0	6.0		
100 gm. of mashed potatoes	3.1	0.5	21.3	
10 gm. of malt extract	0.5	. .	5.5	
In the afternoon:				
1 cup of tea (with zwieback).				
20 gm. of butter	0.15	16.6	0.12	
30 gm. of honey	0.4	. .	22.0	
In the evening:				
250 gm. of rice mush	22.0	8.25	71.0	
In the course of the day:				
75 gm. of zwieback (or toast)	9.0	1.5	63.9	
In the evening at 10 o' clock:				
250 gm. of milk	8.7	9.3	12.0	
10 gm. of cognac (brandy)	. .	. .	. .	7.0
	72.7	62.85	219.64	19.0
Total caloric value	1930 calories.			

The following diet-list is taken from Biedert and Langermann's *Dietetics:*

	Albumin.	Fat.	Carbohydrate.	Calories.
In the morning at 6 o' clock:				
500 gm. of milk, 3 zwieback (30 gm.)	20.6	20.2	45.7	461
In the morning at 8 o'clock:				
Oatmeal soup (made from 20 gm. of oatmeal with meat broth or water), with 15 gm. of meat solution and the yolk of 1 egg	8.5	6.0	14.2	149
or soup made from ¼ of a Timpe soup tablet.				
In the forenoon at 10 o'clock:				
Cream mixture (made from 125 c.c. of cream and 6 gm. of milk-sugar), 40 gm. of toast	7.8	12.9	41.5	322
At noon (12 o'clock):				
(*a*) Leguminose soup (from 20 gm. of leguminose) with 20 gm. of meat solution	10.8	1.8	12.6	113
or soup made from ¼ of a Timpe soup tablet.				
(*b*) "Karthäuserklösse" (made from 100 gm. of rolls, 60 c.c. of milk, 1 part of white of egg, 20 gm. of butter, and 10 gm. of sugar).	12.0	19.5	67.0	506
(*c*) 100 gm. of apple sauce	0.4	. .	17.7	74
In the afternoon at 4 o' clock:				
250 c.c. of milk-water cocoa (from 10 gm. of cocoa, 10 gm. of sugar, 125 c.c. of water), 3 zwieback (30 gm.)	9.2	11.3	38.3	300
In the evening at 7 o'clock:				
(*a*) Milk soup (from 250 c.c. of milk with 10 gm. of flour)	9.5	9.1	19.2	205
(*b*) ¾ groats as mush (from 100 gm. of groats with 500 c.c. of milk and 30 gm. of sugar)	80.5	13.8	97.3	612
	99.3	94.6	353.5	2736

It is unnecessary to give other diet-lists, for any physician with the aid of the tables given in the general part of this work can construct lists that are suitable to each case.

In cases of advanced anemia in which the general nutrition is very much impaired it is best to advise the patients to remain in bed for a long time. If this is done, it is much easier to improve nutrition.

If there is a tendency to atrophy, or if atony has developed, it is necessary to re-establish the tone of the stomach as much as possible. In order to do this we may employ the electric current, or we may perform massage, or we may institute methodic lavage of the stomach. In regard to the best methods of carrying out this treatment I refer to what has been said in preceding chapters.

If there is fermentation, certain antifermentative remedies may be added to the wash-water when lavage is performed. If there is advanced ectasy in addition to atrophy, the propulsion of the ingesta into the intestine is rendered very difficult, and, as a result, the general nutrition of the patient must suffer in a short time. In cases of this kind rectal alimentation may be provisionally employed. The last resort is an operation that will create a new passage between the stomach and the intestine, and render the exit of the ingesta from the stomach into the intestine more easy, and enable the stomach in this way to get rid of its contents more rapidly. The best operation for this purpose is gastro-enterostomy. Even in cases of pyloric stenosis, however, the removal of the obstruction rarely suffices, for the tone of the gastric muscularis is hardly ever restored.

Drugs are seldom indicated in atrophy of the gastric mucosa. Certain symptoms, as anorexia, pain, and diarrhea, may call for medication. The same remedies should be administered here as in any instance where these symptoms develop. Only one method merits particular mention, namely, the administration of hydrochloric acid, pepsin, pancreatin, and similar remedies, that in these cases may replace the lost digestive powers of the stomach.

I can only repeat what I have already said in discussing the treatment of simple chronic gastritis. However useful all these remedies, particularly hydrochloric acid and pepsin, may appear from a theoretical point of view, they do not in reality do what is expected of them. Some investigators, it is true, are full of praise for the powers of hydrochloric acid, but I fail to find any direct evidence of its value even when it is administered in very large doses. Whether or not lavage of the stomach with a 2 per cent. hydrochloric acid solution, and leaving a portion of this solution in the stomach, as advised by Litten and others, is a better method of treatment, and one that yields more satisfactory results, I am unable to say.

ROUND ULCER OF THE STOMACH.

Syn.—*Ulcus Ventriculi Simplex, Ulcus Rotundum Perforans Chronicum, Peptic Ulcer, Perforating Gastric Ulcer, Cruveilhier's Disease.*

A review of the literature shows that the older pathologists described a few cases of round ulcer of the stomach. Only a few isolated case-reports, however, were published in those days. The points of difference between other forms of gastric ulcer, particularly the carcinomatous forms, were unknown. I refer to Lebert[1] for a summary of this older literature. This author has collected the most important investigations on this subject in his contributions to the history and etiology of gastric ulcer.

Cruveilhier[2] was the first to recognize gastric ulcer as a typical well-characterized disease-form. He deserves credit for having furnished the first exact anatomic description of this lesion, and at the same time of having given the first careful description of the clinical symptoms and of many therapeutic measures. Gastric ulcer, therefore, is called Cruveilhier's disease. Following the fundamental investigations of this author, a number of other classical investigations were published. The most important of these is that of Rokitansky,[3] which is based on a large number of cases. Then Jaksch[4] published a series of very careful clinical observations. After the fourth decade of this century, new contributions to this subject were furnished nearly every year. The experimental investigations of Panum, C. I. Bernard, Cohnheim, Daettwyler and Quincke, Silbermann, etc., have contributed much to our knowledge of this subject. Within quite recent years there has been further progress, and it has lately been shown that certain peculiar perversions of gastric secretion are always found in gastric ulcer. This knowledge, it appears to me, gives us a better insight into the genesis of ulcer, and at the same time a more rational basis of treatment.

It would lead us too far to enumerate all the investigations that have been published within the last decade. Such an undertaking, moreover, would be utterly useless. The more important literature on the subject up to the year 1878 can be found in Leube's excellent work on "Diseases of the Stomach," in v. Ziemssen's *Hand-book*. In the following summary I give only the more important investigations that have appeared since that time, and a few of the older reports that I shall have to refer to in the course of my discussion.

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[1] *Berlin. klin. Wochenschr.*, 1876, No. 39, etc.

[2] *Anatomie pathologique*, 1829–1835, vol. i.; *Revue médicale*, 1838; *Arch. gén. de méd.*, 1855.

[3] *Oestreich. Jahrb.*, 1839.

[4] *Prag. Vierteljahrsschr.*, 1843.

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Etiology.—Gastric ulcer is a disease that physicians frequently encounter. It is generally found to be more common in women than in men, but to judge from the different statistics that have been published the relative proportion in men and women varies. In Breslau, Lebert found that the proportion of men to women was as 3–4 to 1; in Zürich he found that more cases occurred in women than in men. Brinton found the proportion of men to women as 2 to 1; Steiner as 11 to 8; and Wollmann found about the same proportion in his cases. Habershon, in 201 cases of ulcer that he examined, found 127 in women and 74 in men. Anderson found only 3 cases in men among 35 cases of ulcer. According to Danziger's statistics from the Würzburg clinic, the proportion of women to men was as 2 to 1.

I have summarized the clinical cases that I observed during the course of many years, and find that 126 cases occurred in men and 134 in women. Great importance, however, should not be attached to these figures, for if I study the cases that occurred during a single year I find some years in which there were more cases of ulcer in women than in men, and others again in which the reverse was the case. In the year 1884, for instance, there were 15 cases of ulcer in men and 8 in women; in 1887 there were 17 in men and 19 in women; and in 1886, 12 in men and 26 in women. This seems to show undoubtedly that chance plays a large rôle. We are certainly not justified in concluding that in any one year ulcer occurs more frequently in men than in women, and that the next year the reverse is the case. Patients enter the hospital for many reasons, and the figures given here are no conclusive index of the frequency of the different forms of the disease. It may be said, however, that ulcer is encountered more frequently in women than in men.

The best way of estimating the frequency of gastric ulcer is to study autopsy statistics. According to Berthold, 262 cases of ulcer were found in the Pathologic Institute of the Berlin Charité in the fifteen years from 1868 to 1882; this means that ulcer or evidence of old ulcers was present in 2.7 per cent. of all the cases that were examined postmortem in that Institute; of these cases, 128 occurred in men and 134 in women.

The figures obtained in the Münich Pathologic Institute are not so high. According to Nolte, ulcer was found in only 1.23 per cent. of all autopsies; of these, 0.8 per cent. were seen in men and 1.8 per cent. in women. Griess, in Kiel, found ulcer in 8.3 per cent.; Ziemssen, in Erlangen, in 4.55 per cent.; Stoll, in Zurich, in 2.16 per cent.; Stark, in Copenhagen, in as many as 13 per cent. Brinton states that 5 out of every 100 autopsies revealed the presence of gastric ulcer. Grünfeld found exceptionally high figures; he succeeded in demonstrating the presence of ulcer scars in 92 out of 450 autopsies (241 women and 209 men)—that is, in 20 per cent.; of the 92 cases, 77 were found in women and 15 in men. Fiedler examined 2200 bodies, and found ulcers or cicatrices in 20 per cent. of female bodies, and in 1.5 per cent. of male bodies. The attempt has been made to explain these variations as due to certain regional differences; in part, however, they must be attributed to the different clinical material that enters different hospitals; I have called attention to this point in another place when discussing mortality statistics.

We know comparatively little in regard to the geographic distribution of ulcer. Reliable statistics that would enable us to formulate a judgment in regard to the frequency of ulcer in different countries cannot be arrived at in hospitals nor in pathologic institutions, but must be gathered by physicians in general practice. No statistics of this kind have ever been published. Gerhardt has repeatedly called attention to the frequent occurrence of ulcer in Thüringia. According to von Sohlern, ulcer is rare in Russia; he attributes this to the fact that more vegetables than meat are eaten in Russia, and that consequently more potassium is introduced with the food, and believes that the rarity of round ulcer in these regions is due to the large proportion of potassium that is consequently present in the blood of the inhabitants. The same is seen in the Rhine region and in the Bavarian Alps. Westphalen contradicted this statement of von Sohlern, and denied that hyperacidity and gastric ulcer are rare in those regions where little meat is eaten, as, for instance, in Russia. He found hypersecretion in 1 out of every 3 or 4 cases of stomach disease that he encountered in his Russian patients. It is also claimed that the peasantry of the province of Oberhessen eat very little meat and live almost exclusively on a vegetarian diet, nevertheless both ulcer and hyperacidity are very frequently encountered in this part of the country.

Age undoubtedly has a certain influence on the development of ulcer. The disease is most frequently encountered between twenty and thirty. According to Danziger's statistics from the clinic of Würzburg, ulcer is most frequently encountered between twenty and thirty, 40.2 per cent. of all cases occurring during these years. The following table represents a summary of 260 cases that I observed myself; it is arranged according to the ages of the patients:

NUMBER OF CASES OF ULCER AT DIFFERENT AGES.

Age.	Men.	Women.	Sum total.
10–20 years	8	35	43
20–30 "	29	62	91
30–40 "	35	22	57
40–50 "	36	11	47
Over 50 "	18	4	22
Sum total	126	134	260

From this table it will be seen that by far the majority of cases occur between twenty and forty. In women the maximum is between twenty and thirty; in men, between thirty and fifty. Isolated cases of gastric ulcer are, however, seen in advanced years. Mortality statistics are, of course, more important than autopsy statistics in determining the age at which ulcer of the stomach is most frequently encountered. If postmortem examinations of very old people reveal the presence of ulcers or cicatrices, it does not at all show at what time during the patient's life the ulcerative process began.

Lebert has also arranged a summary of 252 cases. According to this author, nearly one-half of the cases are found between childhood and the thirtieth year. A large number—that is, about a quarter to a third—in the fourth decennium. Lebert found the greatest mortality between forty and sixty years. According to Habershon, the majority of the 201 cases that he examined in male subjects occurred between forty and forty-five; in female subjects, between twenty and thirty. Gastric ulcer, at all events, is a rare occurrence in childhood. Among the 262 cases of gastric ulcer that were examined postmortem during fifteen years at the Berlin Pathologic Institute no case under ten years was discovered. Here the great majority of the cases were found in subjects that were from twenty-six to thirty years old when they died. The majority of authors declare round gastric ulcer to be a rare occurrence in childhood. According to Kundrat, ulcers about as large as a millet-seed or a lentil, and extending deeply into the tissues, are quite frequently found in children. Kundrat assumes that these ulcers originate from hemorrhages into the mucosa, and argues that the difference between these small and recent sores and round ulcer is quantitative and not qualitative. He even reports the occurrence of little ulcers in children that were only a few days old. Kundrat, however, admits that he has never known chronic ulcers to develop from these little ulcerations in subjects under ten years, and interprets this peculiarity by assuming that acid catarrh is very rare in childhood. Widerhofer agrees with all other authors in stating that gastric ulcer never occurs in childhood. According to Henoch, ulceration of the gastric mucosa is quite frequently found in the newborn. Wertheimer reports a case of ulcer in a child of ten years; Eross, one in a girl of twelve years. Rehn reports a similar case, and collected nine cases of simple ulcer in children from the literature. From all this we may at least assume that round gastric ulcer proper is very rare in childhood. The small erosions that are occasionally seen in the newborn probably have nothing to do with typic gastric ulcer.

The profession or the calling does not exercise any considerable influence on the development of ulcer. We find ulcer in men and women of the highest and the lowest classes, in all professions and callings, in savants, in officials, in mechanics, day-laborers, etc. It is claimed that a few callings predispose particularly to gastric ulcer. It is said, for instance, that cooks develop ulceration of the stomach more frequently than other people because they have to taste food when it is very hot. Decker performed some experiments in order to establish the validity of this theory. He administered small quantities of gruel that was heated to 50° to two dogs. In one animal a hemorrhagic extravasation was found postmortem between the mucosa and the muscularis; in the other dog he found hemorrhage and also a loss of tissue leading to complete destruction of the muscular layer of the stomach. These experiments, however, contribute very little to our knowledge of the etiology of ulcer. No doubt high degrees of temperature can produce hemorrhage, sloughing, and erosion of the gastric mucosa; the muscularis even may be destroyed, as in the second experiment (Decker), but this does not necessarily constitute a lesion that is similar to typic round gastric ulcer. I am, moreover, inclined to disbelieve the statement that cooks furnish so large a proportion of ulcer patients. When we remember that servant girls and cooks form a large proportion of our hospital patients because they are immediately sent to the hospital as soon as they are sick, we need not be surprised to find a relatively large number of cases of ulcer of the stomach in this class. If we remember how many people are engaged in cooking, and consider the relatively small number of these that are afflicted with gastric ulcer, we can hardly say that this disease occurs with greater frequency in these people than in others. It may be true that cooks taste food when it is too hot, and that certain lesions of the gastric mucosa result therefrom, but these lesions are not typic gastric ulcers. In order that the latter condition arise, a number of other things must occur that will be discussed in the chapter on the Pathogenesis of Gastric Ulcer.

Mechanical injuries of the inner wall of the stomach, chemical irritation, etc., may produce lesions of the gastric mucosa, hemorrhages, and similar conditions, in the same way as heat, but all these lesions do not constitute typic gastric ulcer.

Bouveret states that mirror-polishers and metal-workers are particularly predisposed to gastric ulcer. Bernitz claims that workers in porcelain swallow large quantities of sharp particles of dust, and that this may be responsible for gastric ulcer.

Other causes that have been made responsible for gastric ulcer are traumata; for instance, a blow in the region of the stomach. Leube has reported such a case in which gastric symptoms occurred immediately after the trauma. This patient was squeezed against a wall by a wagon in such a way that the shaft pressed against the epigastric region. It was impossible to pull the wagon back for ten minutes; from the time of the accident the patient complained of pain in the stomach. Eight days after the accident vomiting occurred, that was repeated every day thereafter, usually in the afternoon. No blood was ever discovered in the vomit. The pain was limited to a circumscribed area, and was increased by external pressure and by lying on the left side. Under appropriate treatment the symptoms soon disappeared. Leube states that this case was undoubtedly one of ulcer caused by trauma.

Ritter has reported a similar case. The patient was a young, robust individual that had always been perfectly healthy. A few weeks before his admission to the hospital the patient fell from a chair in such a manner that he was thrown against the edge of a table, and struck on his stomach. A few days later he complained of violent pain after eating, and eight days after the accident he vomited blood. Ritter assumes that in this case a hemorrhage occurred under the mucosa of the stomach, causing separation of the mucous membrane from its substratum; in this way nutrition was interfered with, so that it was gradually digested, and thus led to the formation of an ulcer.

Similar cases have been reported by Potain, Bouveret, Derouet, Richardière, Ebstein, and others.

In two cases that Ebstein reported, the disease was caused by lifting a heavy weight. Ebstein assumes that this overexertion caused rupture of some gastric blood-vessel, and that in this way hemorrhagic infiltration of the gastric mucosa occurred that finally led to ulcer.

In order to determine whether traumata of the gastric region can cause an ulcer, Ritter performed a number of animal experiments. Immediately after feeding dogs he administered moderate blows in the region of the stomach, and repeated this procedure several times in a number of animals. Immediately after administering the trauma the animals were killed. In one case a hemorrhage underneath the mucosa was found; in another the mucosa was seen to be separated from the submucosa, and there was hemorrhagic extravasation into the torn submucosa. Ritter argues that in all these instances the peptic action of the gastric juice would very soon have led to an ulcer.

There seems to be no doubt, therefore, that hemorrhages into the gastric mucosa can be caused by a violent blow, a fall on the region of the stomach, etc. It is also certain that under definite conditions there may be a loss of substance, and that ulcer may develop. Ulcers seem to have a predilection for the lesser curvature of the stomach, and it is precisely this portion of the stomach that is usually injured when the stomach is subjected to external violence, particularly if the organ is distended with food or gas. The reason for this is that this portion of the stomach is in close proximity to the unyielding spinal column.

It is probable that trauma, as in the experiments of Ritter, primarily leads to extravasation of blood into the mucosa and a partial tearing of this membrane; but this need not necessarily lead to a round ulcer of the stomach. We know that destruction of the mucosa and erosions that extend deep into the muscularis of the stomach may be cured in animals. This has been positively demonstrated by a number of experiments. This restitution to normal, it appears, may occur in a very short time, and there is a natural tendency in this direction. As a matter of fact, a number of cases have been observed in which symptoms resembling ulcer appeared soon after some trauma, but rapidly disappeared. Slight injuries are undoubtedly frequently sustained, but only in rare instances do we see the development of chronic ulcer. This indicates that other factors are necessary in order to produce a chronic ulcer on the basis of hemorrhagic infiltration of the stomach-wall. I agree with Richardière, who also reports a number of cases of traumatic ulcer, when he states that this disease may progress in two different ways: one group of cases is characterized by very violent symptoms in the beginning, but is rapidly cured; a second group runs a more protracted course, resembling the picture of classic ulcer of the stomach. Richardière believes that these differences are due to variations in the strength and constitution of the gastric juice. If the gastric juice is normal, the wound will heal as rapidly as any other simple wound; if the acidity of the gastric juice is increased, the affection gradually develops into a typic ulcer of the stomach.

Long-continued pressure in the region of the stomach has been considered another causative and predisposing factor in ulcer, belonging to the same class as sudden traumata. Rasmussen in particular has called attention to the effect of lacing on the stomach, and claims that the continuous pressure of corsets may lead to the development of ulcer. He argues that lacing causes pressure upon the stomach, and that in certain portions of the organ atrophy of the gastric mucosa results. This leads to pressure-neurosis, and possibly to ulcer. Lacing undoubtedly exercises an unfavorable effect upon the stomach, for we know that it may produce dislocation of the organ—gastroptosis and enteroptosis. Meinert in particular has shown that this frequently occurs in young women. Lacing alone, however, will never lead to the development of an ulcer as long as other factors that favor this condition are absent.

Relations of Chlorosis and Anemia.—The belief has been prevalent for a long time that chlorosis and anemia are in some way related to ulcer of the stomach. Leube, in his dissertation on diseases of the stomach in Ziemssen's *Hand-Book* of 1878, says: "A weak constitution, chlorosis, and anemia predispose more to ulcer than a vigorous body. Occasionally *homines quadrati*, however, develop this lesion." He does not express an opinion on the inter-relationship between chlorosis and ulcer.

Korczynski and Jaworski report that of 24 cases of ulcer that they examined, all were anemic, that 13 of them had moderate anemia, and that 11 had a waxy complexion. All this, however, does not establish any etiologic relation between chlorosis and anemia and ulcer.

An ulcer that has existed for a long time naturally leads to secondary anemia. The question to be decided is, whether or not chlorosis and anemia favor the development of ulcer. Only such cases could give us an answer to this inquiry in which chlorosis or anemia exists for a long time before symptoms of ulcer develop. We have no statistics in regard to the frequency of this occurrence. Cases, however, are frequently encountered in which ulcer is erroneously taken for chlorosis because ulcer symptoms are absent. Von Hösslin[1] goes so far as to state that chlorosis in the majority of cases is due to gastro-intestinal hemorrhage. He believes that in the majority of cases there are small superficial losses of substance in the gastro-intestinal tract, and that only in the minority of cases are there pronounced ulcers. He also believes that a deficiency of hydrochloric acid in the gastric juice aids the development of ulcer.

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[1] *Münch. med. Wochenschr.*, 1890, No. 14.

Whether or not small superficial solutions of continuity occur in young women as frequently as we must assume if this author is correct, is exceedingly doubtful. Clinically it is established that chlorotic and anemic subjects are more predisposed to ulcer than robust and healthy subjects.

Certain experiments also indicate that a certain relation exists between anemia and chlorosis. Quincke and Daettwyler have found that artificial gastric ulcers are not cured as rapidly in dogs in which anemia has been experimentally established as in dogs in which anemia was not present. They draw the conclusion from these experiments that if anemia is present comparatively slight injuries of the gastric mucosa may lead to the development of ulcer, and that ulcers that already exist heal less rapidly. Silbermann performed another kind of experiment. He produced artificial anemia in animals by the exhibition of hemolytic substances, and found that an artificially created ulcer of the stomach was cured less rapidly under these conditions than in normal animals. None of these investigators has been able to determine by experiments why anemia renders the cure of an ulcer of the stomach more difficult.

Many years ago I called attention to the fact that in chlorosis we frequently find hyperacidity of the gastric juice. This statement was in complete opposition to the view that was generally accepted, namely, that in anemia very little or no hydrochloric acid whatever was secreted. In Grüne's dissertation I had the author collect 19 cases of pure chlorosis, in all of which exceptionally large amounts of hydrochloric acid were found. Other authors, however, have reported different findings. Ritter and Hirsch, for instance, arrived at the conclusion that in chlorosis and anemia there is a reduction of the hydrochloric acid secretion. Lenhartz examined 15 cases of chlorosis and anemia with acute dyspepsia, and 31 cases with chronic dyspepsia, and found varying results; in some of the cases the secretion of hydrochloric acid was increased, in others it was normal, in others it varied, and in still others no hydrochloric acid whatever was found. Ritter and Hirsch unfortunately did not perform their experiments in accordance with the methods that are generally in use, so that their results cannot be compared with those obtained by other authors. Lenhartz's experiments were performed in walking patients, so that the results are ambiguous in this sense. I do not think the investigations performed in such cases are very valuable, chiefly because a great many of the necessary precautions are omitted, and because the different patients cannot be examined as often as necessary. The results reported by Schätzell are in my opinion, therefore, more important. This investigator examined 30 cases of chlorosis in Leube's clinic. He found hyperacidity in 73 per cent. of all cases, normal acidity in 20 per cent., and subacidity in 7 per cent. Cantu also reports the almost constant occurrence of hyperchlorhydria in chlorosis. Oswald has recently published some investigations from my clinic; he examined 21 patients, performed 84 individual tests, and discovered hyperaciditas hydrochlorica in 95 per cent. of all the cases. Bouveret expresses himself in a similar sense, and claims that chlorosis is frequently accompanied by hyperchlorhydria.

We shall see below that hyperchlorhydria does not lead to the development of chronic ulcer, but that it plays an important rôle in preventing its cure. If we concede that hyperchlorhydria is present in the majority of cases of chlorosis, this alone may explain why ulcer of the stomach and chlorosis are so frequently found together.

Other Causes.—Some authors state that alcoholic excesses may lead to the development of ulcer, and de la Tourette goes so far as to claim that alcoholism is the most important etiologic factor. Mathieu believes that the hyperacidity of the gastric juice that is constantly found in alcoholics is the connecting link between alcoholism and the development of ulcer. His premises, however, to judge from my investigations, are not correct. It is true that drunkards are frequently afflicted with catarrh; that the stomachs of these subjects are frequently covered with small erosions; but I do not believe that hyperacidity nor ulcer is particularly frequent in these cases; nor do I agree with de la Tourette when he claims that hysteria is another frequent cause of ulcer. It is possible that this author confuses ulcer with another condition of the stomach that Sticker[1] has recently described, namely, a hyperesthetic condition of that organ that is quite frequently found in hysteric subjects. I refer to Sticker's dissertation for the typical stigmata of this hysteric form of hyperesthesia, and will mention his work again in the section on Differential Diagnosis.

The concurrence of burns of the skin and of gastric and duodenal ulcers is particularly interesting. In cases of severe burns duodenal ulcers are most frequently encountered, but a number of cases of ulcer of the stomach have also been reported (Wilks, Pitt, and others).

Ebstein reports the occurrence together of gastric ulcer and trichinosis. His patient was a young girl who had eaten large quantities of meat containing trichinæ. The autopsy revealed the presence of five round ulcers in the pyloric region of the stomach.

Certain toxic substances and certain drugs may erode the gastric mucosa and lead to the formation of ulcers. As a rule, however, the lesions in these cases are not gastric ulcers proper, but more or less extensive destructions of the gastric mucous membrane that may either be superficial or may extend into the deeper tissues. Tuberculosis and syphilis may cause similar lesions, but they are not directly related to ulcer.

Several authors have attributed an important rôle in the etiology of ulcer to micro-organisms, and claim that a certain number of peptic ulcers of the stomach are of mycotic origin. Boettcher, for instance, found colonies of micrococci at the margin and the base of ulcers of the stomach. Koerte, however, questioned the validity of this finding, for he showed that mycosis might develop secondarily in a fully developed ulcer. Recently Letulle called attention to the possibility that ulcer may be due to infection. He found bacteria in the stools of a man who was suffering from dysentery, and who developed all the symptoms of gastric ulcer. An inoculation of pure cultures of these bacteria into a guinea-pig produced specific erosions and ulcers of the stomach. The same author also saw 4 cases of gastric ulcer that developed in the course of a suppurative process. In one case of recent ulceration of the stomach that occurred during the course of puerperal septicemia numerous streptococci were found, both in the veins of the submucosa and in the veins of the uterus. When he injected these streptococci into guinea-pigs, he saw the development of gastric ulcers. According to Letulle, this infectious form of gastric ulcer may originate in two ways: in rare instances it may proceed from the surface of the mucosa, provided micro-organisms find a nidus there for their development; or it may follow embolisation which leads to necrosis of the mucous lining of the stomach and erosion of the mucosa by the gastric juice.

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[1] "Beiträge zur Hysterie: hysterischer Magenschmerz, hysterische Athmungsstörungen," *Zeitschr. f. klin. Med.*, vol. xxx., p. 61.

Nauwerck, in order to demonstrate by anatomic methods that many peptic ulcers may be caused by mycotic influences, studied hemorrhagic erosions of the stomach with particular care. He arrived at the conclusion that the majority of hemorrhagic erosions are due to peptic solution of the gastric mucosa after it had become necrotic as a result of hemorrhagic infiltration, but that a certain proportion of the erosions are due to mycotic necrosis of the gastric mucosa.

Bouveret is undoubtedly right when he says that round gastric ulcer proper is different from these ecchymoses, infarcts, and ulcerations of the gastric mucosa that can be produced by experimental infection. Bouveret also calls attention to the fact that it has never been clinically demonstrated that gastric ulcers frequently follow infectious diseases.

Talma attributes a particularly important rôle in the etiology of ulcer to spasmodic contractions of the stomach. He applied faradic electricity to the left pneumogastric nerve of rabbits for prolonged periods of time; at the same time he allowed solutions of hydrochloric acid to flow into the stomach so that a secretion of gastric juice was induced. In all these experiments ulcers developed. Talma believes that the frequent occurrence of ulcers in the pyloric portion of the stomach—that is, in the most muscular part of the organ—supports his theory.

Schiff and Ebstein caused gastric hemorrhage, hemorrhagic infiltration, and ulceration of the gastric mucosa in animals by inflicting injuries to certain portions of the brain—as, for instance, the anterior corpora quadrigemina, by incision into one half of the medulla oblongata, and by injuries inflicted upon certain portions of the spinal cord. No corresponding observations have so far been made in human subjects. This is essentially all we know in regard to the factors that cause gastric ulcer and predispose to it.

I will now proceed to the discussion of its pathogenesis. The facts that have been discovered so far are not at all satisfactory, and furnish only an incomplete examination of the pathogenesis of gastric ulcer.

My reason for entering into the details of this question is that it is not only theoretically, but also practically and therapeutically, interesting. Only when we know what conditions lead to the formation of an ulcer, what factors impede its cure, and what elements favor its persistence, may we expect to cure this lesion by rational procedures.

Pathogenesis.—The fact that that form of ulcer that we call *ulcus ventriculi rotundum* occurs only in the stomach, and its immediate vicinity has given rise to the theory that it is primarily due to a corroding action of the gastric juice. For this reason, too, it has been called *ulcus pepticum.* The logical sequence of this view would be to inquire why the gastric mucosa does not digest itself under normal conditions. Until recently the view was held almost universally that only the most superficial portion of the gastric mucosa could possibly be digested; for it was found that those portions of the gastric mucosa that corresponded to the fundus of the gastric tubules were alkaline in reaction, and could consequently not be digested by gastric juice. Leube[1] says: "In order that destruction of the deeper layers of the gastric mucosa can occur, the normal alkaline reaction of these parts must first be neutralized by the gastric juice. This can occur only under two conditions—namely, either when the gastric juice is abnormally acid or when the alkalinity of the walls of the stomach is reduced."

Under normal conditions the gastric juice does not destroy the mucosa of the stomach during life. As soon as death occurs and the circulation in these parts stops, the digestion of the gastric mucosa begins; here the gastric juice digests the walls of the stomach as it would any other dead tissue. If considerable quantities of gastric juice are present in the stomach after death, it digests the stomach mucosa just as it does the ingesta. The degree of this cadaveric softening of the stomach varies according to the quantity and constitution of the gastric juice, according to the temperature of the body and the state of the mucosa itself. Those portions of the stomach in which the gastric contents accumulate are particularly predisposed to postmortem softening. If the body is lying on the back, the posterior wall of the stomach will, therefore, be destroyed first. The factors we have enumerated above will determine whether we see so-called brown or gelatinous softening; both conditions, we must remember, are due to postmortem changes.

According to this view, the reason why the gastric juice does not normally digest the walls of the stomach during life is that these walls are continuously flooded with alkaline blood, and that, consequently, the gastric juice is always neutralized. If the circulation is impeded in any one circumscribed portion of the stomach-wall, autodigestion of this portion occurs, because no more alkaline blood can flow through it. In this way it is believed an ulcer may originate.

This view is very seductive on first sight, and was generally accepted, and in a short time replaced the older view of Hunter. The latter believed that a certain vital principle inherent in the parts protected the walls of the stomach from autodigestion. Hunter says: "If a person would put his living hand into the stomach of an animal while digestion is going on, the hand would not be digested; if, however, the hand were cut off and placed into the stomach, it would be digested." Hunter's theory was robbed of its main support when Claude Bernard showed that the leg of a living frog could be held in the stomach of a dog, and was in part digested after three-quarters of an hour. The same investigator injected gastric juice underneath healthy skin and caused far-reaching destruction of tissue. Claude Bernard drew the following conclusions from these experiments: He argued that life *per se* does not protect the stomach from autodigestion, and that, as a matter of fact, parts of the stomach are in part digested during life, chiefly, of course, the superficial layers of the epithelium and the mucus that is excreted there. The deeper layers of the stomach are protected from the dissolving action of the gastric juice by the rapid growth of the epithelial cells, which proceeds so quickly that the uppermost layer is replaced as soon as it is removed by digestion.

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[1] *Ziemssen's Handbuch d. spec. Pathol. u. Therap.*, 1878, vol. vii., pt. ii., p. 93.

Pavy was able to corroborate Claude Bernard's results. He experimented with the tip of a rabbit's ear, and found that it, too, was digested like the leg of a living frog when placed into gastric juice. He did not, however, agree with Claude Bernard in regard to the protective action of the superficial epithelium of the stomach, for he removed a large portion of the gastric mucosa and found that the portions of the gastric wall that were situated immediately beneath the denuded area remained undigested as long as the circulation of the parts was undisturbed. If, however, the circulation was interrupted, even though the epithelium remained intact, autodigestion of the stomach-wall occurred. He also saw autodigestion in cases where the circulation and the epithelium both remained intact, but in which an excessive quantity of acid was introduced into the stomach.

From these observations Pavy drew the conclusion that the regeneration of epithelium did not protect the stomach from autodigestion, but that the continuous circulation of alkaline blood through the mucosa neutralized all the hydrochloric acid that permeated into the mucosa, and in this way prevented destruction of the deeper layers of the stomach-wall. Pavy is further inclined to attribute the digestion of the leg of the frog and the tip of the rabbit's ear, even though circulation was maintained in these parts, to the small amount of blood that circulates through these tissues.

The theory was prevalent for a long time that disturbances of circulation in circumscribed areas of the stomach might lead to round ulcer. As early as 1800 Morin, in his dissertation on "Perforation of the Stomach," argued that stasis in the blood-vessels and changes in the circulation and the nutrition of the stomach-wall might lead to chronic ulcer. Virchow, in particular, demonstrated the hemorrhagic-necrotic origin of ulcer of the stomach and was responsible for the universal acceptance of this view. As early as 1855 Virchow called attention to the importance of circulatory disturbances in the etiology of ulcer. According to this investigator, trophic disturbances of the vessel-walls, aneurysmatic and varicose dilatations, and, in particular, thromboses and emboli, are the most prolific causes of ulcers. Other authors (Key) attribute the origin of gastric ulcer to impediments in the venous circulation caused by strong contractions of the gastric musculature, transitory venous stasis, and resulting hemorrhage. Klebs thinks that local ischemia following spastic contractions of arteries should be made responsible. Openchowski attributes an important rôle to hyaline degeneration of the walls of the smallest blood-vessels ; still other authors are inclined to attach significance to hemorrhagic erosions of the stomach-wall, and to consider them as the starting-point of gastric ulceration. However different, therefore, the views of all these investigators are in regard to the special kind of circulatory disturbance, all agree on this, that interruptions in the circulation of the blood in circumscribed districts of the wall of the stomach constitute the chief cause of gastric ulcer. They all believe that, as a result of these circulatory disturbances, an insufficient quantity of blood flows through the affected area ; that, consequently, the normal alkalescence of the stomach-wall is reduced, and that these portions, therefore, are subjected to autodigestion by the hydrochloric acid of the stomach.

Another theory of gastric ulcer was promulgated soon afterward, namely, that an abnormal increase in the acidity of the gastric juice could cause ulcer of the stomach even though the alkalescence of the gastric mucosa remained normal. Claude Bernard's experiments seemed to harmonize with this view. Leube[1] believed that an abnormal degree of acidity of the gastric juice could undoubtedly cause erosion of the whole wall of the stomach, or at least of a considerable portion of the wall, if the gastric mucosa remained in contact with the acid gastric content for a prolonged period of time, and that this could occur even though the surface of the mucous membrane of the stomach was perfectly normal. He thinks, therefore, that those who are inclined to attribute the formation of gastric ulcers to an increased acidity of the gastric juice must also concede that a small portion of the stomach-wall may at times be exposed to an abnormal quantity of acid. He argues further that such a possibility is conceivable, but that its validity has never been experimentally demonstrated ; that if the gastric juice is abnormally acid and comes in contact with some portion of the stomach-wall that has been injured in some way, the exposed ends of the arteries are irritated and contract, so that anemia of the injured portion develops. Leube, therefore, believes in the coincidence of two elements that lead to erosion,—namely, anemia and temporary abnormal acidity of the gastric juice,—and claims that both these factors must be present in order that chronic ulcer of the stomach may develop.

This point of view was maintained until quite recently. Even today we must concede that the above theory seems to be correct, for we know that the two main factors that lead to the development of chronic ulcer of the stomach are interruption of the circulation in a circumscribed portion of the stomach-wall, causing local anemia, and abnormal acidity of the gastric juice. I am in doubt whether the other view,—namely, that the flooding of the stomach-wall with alkaline blood and the neutralization of the hydrochloric acid of the stomach protect the organ from self-digestion,—is universally accepted to-day.

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[1] *Loc. cit.*

Both anatomic and experimental facts demonstrate that circulatory disturbances may lead to ulcer. Diseased blood-vessels, venous thrombi, occluded vessels, etc., have been found in the vicinity of gastric ulcers; ulcers have also been seen to develop from hemorrhagic infiltrations of the mucosa. Cohnheim is undoubtedly right when he says that disease of blood-vessels, thromboses, emboli, etc., can hardly be considered a cause of round ulcer, at least not from a pathologic-anatomic point of view, because this disease is chiefly a condition found in the early years of life—that is, at a time when a pronounced immunity against arterial disease seems to exist. He also states that in the majority of cases of ulcer valvular lesions of the heart are absent. This is all true, but in ulcer of the stomach we are not dealing with lesions of the heart nor with arteriosclerosis, but with a variety of external agencies that act, we may say, by chance, and lead to occlusion of blood-vessels, to anemia, or to circumscribed hemorrhages.

The effect of circulatory disturbances on the development of ulcers of the stomach has also been studied by animal experiments. I need but recall the well-known experiments of Panum, who succeeded in causing hemorrhagic infarcts and ulcers of the gastric mucosa by injecting an emulsion of wax particles into the central portion of the femoral artery in dogs. L. Müller produced hemorrhagic erosions and ulcers of the gastric mucosa in rabbits by artificial occlusion of the portal vein and of some of the large veins of the stomach. The experiments of Cohnheim are particularly significant, for this investigator succeeded in producing ulcers that were analogous to gastric ulcers seen in human subjects by injecting a suspension of chromate of lead into one of the splenic or gastric arteries of dogs. The margin of these experimental ulcers was sharply defined; the base was clear. Some of the animals that he experimented on remained alive for several weeks. The ulcer was usually completely cured by the end of the third week. Körte produced ulceration of the stomach-wall by inflicting different injuries—by eroding the stomach-wall, by burning it, by crushing it. Talma caused gastric ulcer by increasing the tension of the stomach-wall; he ligated the upper and lower orifices of the stomach in rabbits, and in this way produced hemorrhages into the mucosa and typical ulcer of the stomach. He also produced ulcers by causing arterial anemia by faradic irritation. We see, therefore, that postmortem findings and experimental research teach us that interruption of the circulation in any circumscribed area of the gastric wall may lead to the formation of ulcers, and that regular and uninterrupted circulation of the blood is the best protection against autodigestion.

At the same time these facts do not decide the question whether the circulation of the blood and the normal nutrition of the gastric wall are the chief protecting factors against self-digestion, or whether it is the alkaline reaction of the blood that neutralizes the acid of the gastric juice. Until quite recently it was believed that interruption of the blood-current in a circumscribed area led to a reduction of the alkaline reaction of those parts or a complete loss of alkaline reaction. Only under these conditions, therefore, it was argued, could acid gastric juice penetrate the deeper layers of the stomach-wall and lead to ulcerative destruction of tissue.

Many years ago I raised my voice against this theory. I have always maintained that the reason why circumscribed areas of the stomach are digested and an ulcer is formed as soon as the circulation is interrupted, is not that the reaction of the tissues becomes acid instead of neutral, but that trophic changes occur; that the tissues are not sufficiently nourished, and consequently die. Dead tissue is naturally digested by the gastric juice. We know that gastric juice does not destroy the delicate layers of epithelium during life; as soon, however, as life ceases, the gastric juice acts on the dead tissue in the same way as it acts on the ingesta. Autodigestion of the stomach during life is impossible.

Even though alkaline blood circulates through the tissues, this does not demonstrate by any means that the cells themselves are saturated with the alkaline fluid. Marchand is justified in asking the question, "Why does the secreting cell not digest itself? It must certainly be acid, for it produces an acid secretion." The main reason why the stomach does not digest itself is unquestionably the vital resisting power of the tissues. Living tissue reacts differently to chemical action than dead tissue; it may be said to possess definite resisting powers against such agencies.

I must oppose another view, namely, that the mucous lining of the stomach is acid only in its most superficial portions, where it is in contact with the acid gastric juice, and that it is alkaline in all the other parts that are bathed in alkaline blood. A number of years ago Edinger carried on some investigations in my laboratory on the reaction of the living gastric mucosa, and found that the gastric mucosa is not only acid near its surface, but throughout its whole extent. If this finding can be corroborated, the dictum that it is the alkaline reaction of the gastric mucosa that protects it from autodigestion must collapse; also the argument that circulatory disturbances in the stomach-wall lead to erosion of the tissues because the acid of the gastric juice is no longer neutralized. The only explanation that remains is that an interruption of the circulation of blood through any given part of the stomach interferes with the nutrition of the tissues, and in this way destroys the power of the parts to resist the digestive action of the gastric juice.

Other arguments may be adduced against the view that Pavy originally expressed, and that is almost universally accepted, namely, that the alkalescence of the blood protects the stomach from the action of the gastric juice. The alkalescence of the blood, in the first place, is so small that it can hardly be considered in relation to the acidity of the gastric juice. In the second place, we frequently see typical ulcers in the intestinal wall in places where a fluid is found that can digest albumin but is alkaline in reaction. If the gastric juice is really alkalinized by the blood, there is no reason why the secretion of the glands that is poured into the stomach should not be neutralized in the first place. If we accept the theory that the acid that enters the stomach-wall from the surface of the stomach—that is, that invades the tissues—is neutralized, there is no reason to believe that the gastric juice that passes through the tissues outward should not also be neutralized in transit.

According to the theory of neutralization, the gastric mucosa is simply a membrane that permits the diffusion of substances in solution. The question arises, Does an equalization between gastric juice and blood alkali occur in the living stomach, and does this neutralization follow the laws of diffusion? Sehrwald has performed a number of experiments that seem to contradict this view: "Whatever diffusion may occur during life is too slight to maintain a neutral reaction of the gastric mucosa, and in this way to protect it from autodigestion. Other factors must be active during life, and these are undoubtedly dependent on the life of the cell, for they are no longer active after death occurs." Sehrwald argues correctly that diffusion of alkali and acid cannot possibly occur during life, as it occurs in a dead membrane, for otherwise the hydrochloric acid of the stomach would be neutralized and peptic digestion would be rendered impossible. If diffusion really occurred in the same way as in any other membrane, the acidity of the gastric juice would remain so low that the time of digestion would be immeasurably protracted or the gastric glands would have to work very much harder, because so large a portion of their labor would be immediately annulled. No one will believe that a portion of the acid of the gastric juice is produced only that it may at once be neutralized by the alkali of the blood. This, however, is what the adherents of the diffusion theory seem to believe. The protective power of the blood is not inherent in the alkali it contains, but in its property to nourish the parts. It is the power of the blood to nourish the cell that protects the tissues against the action of the gastric juice, or, better, of the hydrochloric acid contained in the gastric juice, and not its alkaline reaction.

Claude Bernard's experiment with the thigh of a living frog, previously described, seems to contradict this view, for here the circulation is carried on in a normal manner, but the thigh, nevertheless, dies when introduced into the stomach of a dog. It might appear that here there is, in reality, a disproportion between the acidity of the stomach and the alkalinity of the blood. In this case, however, other factors undoubtedly play a rôle. We know, for instance, that the cells of cold-blooded animals die very rapidly when they are forced to exist at the temperature of warm-blooded animals, and as soon as cells die they are, naturally, digested. Pavy also has called attention to the slight vascularity of the frog's thigh and the rabbit's ear.

All the different observations that we have chronicled do not support the old view that the alkalinity of the deeper layers of the mucosa prevents autodigestion of those parts by gastric juice. Samelson has performed still other experiments that also contradict this view. He introduced large quantities of different acids into the stomach of animals, but never saw autodigestion as long as the blood could circulate freely through the stomach-walls. Even when he injected acids into the blood the stomach remained intact.

In support of the view that the alkalinity of the blood prevents the autodigestion of the stomach-walls by the acid of the gastric juice, the relative frequency with which ulcer is seen in chlorotic and anemic subjects might be adduced. As a matter of fact, von Jaksch has demonstrated that the alkalinity of the blood is frequently considerably reduced in chlorotic subjects; at the same time we are not justified in drawing the conclusion from this that diminished alkalescence of the blood is responsible for the frequent occurrence of ulcer in chlorosis, for we should have to assume that the same tendency to ulcer formation existed in other diseases in which the alkalescence of the blood is considerably reduced. This, however, is not the case. It is true that anemic conditions render healing of the ulcer more difficult. We need only recall the well-known experiments of Quincke and Daettwyler, who showed that the withdrawal of large quantities of blood rendered the healing of artificially produced gastric ulcers more difficult in dogs. This, of course, does not show that the majority of gastric ulcers are due to alterations of the blood. Silbermann's experiments show that artificial gastric ulcers do not heal so readily if there is anemia. He injected hemoglobin or hemolytic substances into animals, and in this way produced hemoglobinuria and anemia; then he produced artificial gastric ulcers, and demonstrated that these lesions did not heal so rapidly in the anemic animals as in the normal ones. All this, however, fails utterly to explain the primary cause of ulcers. We certainly see a large number of anemic and chlorotic subjects who are not afflicted with gastric ulcer. I think we may consider it established that the connection between chlorosis and ulcer is not the peculiar condition of the blood-vessels that Virchow has described in chlorosis. Ziemssen, it is true, attributes an important rôle to this peculiarity, and claims that ulcers are due to the narrowness of the blood-vessels and to premature fatty degeneration of vessel-walls in chlorotic girls. He argues that this condition of the blood-vessels leads to circumscribed hemorrhagic infiltrations and similar conditions of the stomach-wall as soon as any injury, however slight, is inflicted upon the parts. One condition that is frequently seen in chlorosis may possibly be concerned in this matter, namely, hyperchlorhydria.

From all these facts it appears to me we must conclude that the fact that the stomach does not digest itself normally is not due to the neutralization of the acid of the gastric juice by the alkaline blood; but is due, undoubtedly, to the vital resisting power of the tissues. As long as the stomach is normally nourished, and as long as a sufficient quantity of blood circulates through its walls, it will not be digested. An increase in hydrochloric acid alone is not, moreover, sufficient to cause an ulcer; this we learn from simple clinical observation. If the alkalescence of the blood protected the stomach from autodigestion by neutralizing the acid, a great decrease in the alkalescence of the blood should lead to an increase in the acidity of the gastric juice and to self-digestion of the stomach. In reality, however, we frequently find that subjects who suffer from transitory or permanent hyperaciditas hydrochlorica never develop symptoms of ulcer. We see, therefore, that no change in the relation between the acidity of the stomach-contents and the alkalinity of the blood is able to cause ulcer.

It appears to me that many of the authors who have tried to explain the origin of gastric ulcer have not adhered strictly to the facts of the case. An interruption of the circulation in any circumscribed area certainly leads to the formation of an ulcer, whatever the cause of this circulatory disturbance may be. Whenever an insufficient quantity of blood flows through a certain area of the stomach-wall this area must die, just as a toe, for instance, dies when its afferent blood-vessels become occluded.

The rôle of disturbances of circulation in the causation of ulcer has been established beyond doubt by numerous experiments (Panum, Cohnheim, Daettwyler, Körte, Talma, and others). Circulatory disturbances are always the primary factor that lead to the death of a portion of the stomach-wall; the dead piece is, of course, immediately digested by the gastric juice. It would be surprising were this not the case—if the stomach did not digest this dead tissue as it digests any other dead tissue. It is a peculiar fact, however, that the round ulcers of the stomach that are found in human subjects persist with such obstinacy and do not have a tendency to heal. Cohnheim directs attention to the fact that the origin of ulcers is no more obscure than their obstinate resistance to all attempts at cure. All investigators seem to agree that injuries to the gastric mucosa inflicted artificially in animals—for instance, artificial erosions, burns, and crushing of the tissues—heal rapidly. Although Cohnheim, in his experiments, succeeded in producing very large and very deep ulcers, all these lesions healed spontaneously within three weeks. The same observation is reported by all other investigators. In man we see similiar conditions. It frequently happens that pieces of the gastric mucosa are torn off during lavage; Leube, Wiessner, Ziemssen, Schliep, and others have chronicled such accidents. I have personally frequently seen this accident when others were performing lavage, but in no instance have symptoms analogous to those produced by round ulcer been observed. This seems to indicate that defects of this character rapidly heal in human subjects like those that are artificially produced in animals, and that no reactive symptoms are observed during this process of healing. From all this we must assume that in round ulcer of the stomach certain factors obtain that render a cure of this lesion difficult.

Matthes has performed a number of experiments that throw much light on this subject. In the first place, this author pointed out that the mucous lining of the stomach covers a much larger surface than all the other layers of the stomach-wall; this is indicated by the tendency of the mucosa to form folds. If a piece of gastric mucosa is removed in an animal, the mucous lining immediately laps over into the defect, and may even completely cover up any small solutions of continuity or may reduce the size of larger ones to a considerable extent. But even if sufficiently large defects are created artificially, the muscularis becomes exposed, comes in contact with the gastric juice, and contracts very energetically, so that it presses the margins of the mucosa together. The edges of the wound are frequently so closely approximated that it is difficult to find the lesion. In order to prevent this action of the muscularis, Matthes placed flat rings of glass on the serosa of the stomach and attached them to the organ by a number of sutures that passed through the muscularis. When he removed a piece of mucosa that corresponded to the lumen of a ring, he succeeded in keeping the wound open; but even this lesion healed within a short time without injury to the muscularis. When Matthes, however, produced hyperacidity or hyperkrinia in dogs in which he had performed such an operation, healing was greatly retarded. In one of the dogs that Matthes operated on he introduced 350 c.c. of 0.5 per cent. solution of hydrochloric acid through the stomach-tube. The dog was killed after four weeks and an open ulcer was found. According to Matthes, the acidity of the stomach-contents is the only important pathologic factor. [Adolph Schmidt[1] has recently called attention to this well-known contraction of the stomach-walls by means of which any lesion of the mucosa is covered over and shut off from the cavity of the stomach by the overlapping of the mucosa. He believes that in cases of trauma in which the injury is not covered by the folding mucosa an ulcer is likely to develop, provided there is active gastric juice present. He attributes the predilection of ventricular ulcer for the lesser curvature and pyloric region to the fact that the folding over of the mucous membrane, and, therefore, the protection of any lesion, is less perfect at this point. In other words, according to Schmidt, a cause of gastric ulcer is to be found in the absence of the natural power to cover traumatic defects accidentally present in the mucous membrane of the stomach.—Ed.] These experimental observations correspond with clinical facts. I have already stated that many authors attribute gastric ulcers to an increased acidity of the gastric juice as frequently as other authors attribute it to a decreased alkalinity of the blood; I have also mentioned that it is not the occurrence of the ulcer that is peculiar, but merely its tenacity. We assumed *a priori* that the cause of this tenacity is hyperacidity of the stomach-contents, but were unable to prove this proposition. Many authors believe that this acid dyspepsia is not only characterized by an increase of hydrochloric acid, but also by an increase in abnormal fermentation products, as butyric acid, lactic acid, and acetic acid. Until quite recently nothing was known of hyperchlorhydria. As early as 1885 I examined a number of cases of gastric ulcer, and determined the amount of gastric secretion; I found that the gastric juice always contained excessive hydrochloric acid. Soon afterward von der Velden reported 3 cases of ulcer in which he also found a considerable increase in the hydrochloric acid. I have repeated my analyses of stomach-contents in many cases of ulcer, and have succeeded in showing high, or at least increased, values for hydrochloric acid in nearly all cases. I will refer to isolated cases in which the opposite result was obtained in another place—namely, in the section on the Symptomatology. It is generally recognized nowadays that hyperchlorhydria is present in the great majority of cases of ulcer. The absence of this condition must be considered the exception: its presence, the rule. We must assume, therefore, that a close relationship exists between the ulcerative process and the increased production of hydrochloric acid during the act of digestion.

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[1] *German Cong. of Internal Med.*, 1902.

We might imagine, as I originally proposed, that hyperchlorhydria is the primary event and ulcer the secondary one, but some authors have assumed the contrary. Ewald, for instance, considers the reverse to be just as probable. He argues that in individuals who are predisposed to hyperirritability of the secretory nerves of the stomach any injury of the mucosa might lead to hyperchlorhydria and later to typical ulcer. I do not deny the possibility of such an origin of ulcer, but there is one objection to this hypothesis—namely, that a disposition to abnormal irritability of the nerves of secretion must be assumed. Why such a disposition should become manifest in one case in which the gastric mucosa is injured, and not in another, is altogether unexplained. If we assume that the injury and the ulcer are one, I do not see why so many cases of defects of the mucous membrane, of injuries, of hemorrhagic erosions, of circumscribed hemorrhages into the mucosa, run their course without producing any symptoms in particular, and without leading to ulcer, whereas in other cases the same injurious agencies lead to deep ulcerations. The view that ulcer leads to hyperchlorhydria only when it attains a certain size, that, in other words, hyperchlorhydria is an irritative symptom due to ulcer, is contradicted by animal experiments.

Cohnheim and Matthes showed that very large and deep lesions with much loss of substance healed in animals within a very short time. Matthes showed, however, that this restitution to normal was considerably retarded if hyperchlorhydria was produced artificially. The fact that these artificial wounds healed spontaneously and with such rapidity showed that hyperchlorhydria never occurred spontaneously, even though the defects were very large. In the majority of ulcer cases we are probably dealing with small lesions that gradually enlarge. How many times have "artists" performed the trick of swallowing knives and other sharp articles without producing ulcer of the stomach! We must assume that in this trick injuries of the gastric mucosa are unavoidable. Why should these wounds not produce hyperchlorhydria, while in other cases injuries that are certainly less considerable cause the development of ulcerations with hyperchlorhydria? Dr. Marcet[1] reports a very interesting case. His patient was a sailor who frequently performed the trick of swallowing knives; on postmortem examination some thirty knife-blades were found in his stomach, but no evidence of recent or old ulcers. Fricker[2] has quite recently reported another case that is still more striking; here thirty-seven foreign bodies, including one key, two teaspoons, one fork, wire nails, hair-pins, sewing-needles, pieces of glass, etc., were found in the stomach. After the removal of all these bodies the patient rapidly recovered. Why did ulcer not develop in this case, in which the mucous membrane of the stomach was undoubtedly injured?

If traumata of this kind are unable to produce round ulcer in a healthy stomach, we must assume that some particular disposition must exist in those cases in which ulcer does develop. We can hardly assume that this disposition develops at the moment when the injury is inflicted, for we frequently see hyperchlorhydria without ulcer, and occasionally we see that hyperchlorhydria persists or returns even after the ulcer has healed. The tendency to recurrence which we so frequently see in ulcer cases is probably due to the persistence of this hyperchlorhydria.

I am of the opinion that the relation between ulcer and hyperchlorhydria is such that hyperchlorhydria is the primary event. This condition is manifested by an increased production of hydrochloric acid as soon as normal irritants, like food, stimulate the gastric mucosa. If the gastric mucosa is injured in a subject whose secretory nerves are abnormally irritable, whose gastric contents are always hyperacid, the erosion or injury of the mucosa does not heal so rapidly as in a healthy subject. After each meal the hyperchlorhydria returns; the cure of the small lesion is, therefore, prevented just as in the animal experiments of Matthes that we have described above. An injury, therefore, that leads to hemorrhagic erosion or to loss of substance may heal rapidly in one case and may lead to an ulcer in another. D. Gerhardt and Nauwerck have recently demonstrated by anatomic examinations that hemorrhagic erosions may develop into corroding ulcers.

According to D. Gerhardt, small ulcers that resemble hemorrhagic erosions may be caused by swelling and ruptures of follicles. Different authors do not agree in regard to the frequency with which hemorrhagic erosions lead to ulcer; some clinicians consider this to be a frequent occurrence; others, as Langerhans, consider it to be rare. According to the latter author, erosions and round ulcers are not related in regard to their origin, their location, or their form. Langerhans, therefore, advises separating the two conditions more strictly. Other authors, again, as Nauwerck, argue that the two are closely related.

From a clinical point of view, there is no doubt that ordinarily a hemorrhagic infiltration of the mucosa is the primary event, and that this condition secondarily leads to necrosis. This infiltrated necrotic tissue then undergoes peptic solution. The extent of the hemorrhagic infiltration, the condition of the gastric juice, and other factors will determine whether a superficial erosion develops that heals rapidly, or whether a deep ulceration is produced. Nauwerck has recently demonstrated in a number of cases that the reverse may be possible—namely, that there may be hemorrhagic erosions in which the extravasation of blood is the secondary event, and in which the erosion itself is due to a mycotic necrosis of the gastric mucosa.

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[1] *Med.-Chirurg. Trans.*, vol. ii., p. 72.

[2] *Deutsch. med. Wochenschr.*, 1897, No. 4.

Hypersecretion can, of course, act in the same way as hyperchlorhydria, and may impede the healing of an injury to the gastric mucosa and favor the extension of an ulcer. Strictly speaking, an ulcer of the stomach is, therefore, not an ulcer in the proper sense of the word,—that is, an ulcerative process complicated by pus-formation,—but merely a defect and a necrosis.

In conclusion I repeat that it is not so wonderful that ulcers are formed in the stomach, as that round ulcers of the stomach are so difficult to cure in healthy persons and in normal animals, whereas ordinary injuries of the gastric mucosa heal with such rapidity. The reason for this is nothing more than abnormal irritability of the secretory organs of the stomach that manifests itself in hyperchlorhydria. That this is the case, has been fully demonstrated by the clinical and experimental results we have described above.

The experiments of Koch and Ewald correspond with this view. These investigators produced gastric hemorrhages in animals according to the method of Schiffm—namely, by severing the spinal column ; then they introduced a comparatively strong hydrochloric acid solution (5 pro mille) into the stomach, and in this way caused the development of deep ulcers. Ebstein has performed another series of experiments that are exceedingly interesting, but the results are not well understood. They were as follows : He inflicted some injury to the anterior corpora quadrigemina, and in this way brought about gastric hemorrhages with corroding ulcers and even perforation of the stomach-wall.

It is not established whether or not this operative procedure led to the overproduction of hydrochloric acid, although many authors are inclined to this belief. At all events, we must attribute an important part to hyperchlorhydria both in the production of ulceration and in its further development. Hyperchlorhydria, moreover, in the light of our present knowledge, must be made responsible for the obstinate character of round ulcer of the stomach.

In order to do justice to all writers on this subject, I must mention briefly the theory that has lately been promulgated by Stockton. Stockton believes that in addition to the different dyscrasias—tuberculosis, syphilis, pyemia, and scurvy—that can all affect the gastric mucosa, and in addition to the decreased alkalescence of the blood or hypersecretion, certain other unknown forces must act on the stomach. This, he argues, is the only explanation for the fact that gastric ulcer is seen at a certain age,—namely, in the early years of life,—and that it occurs more frequently in women than in men. Stockton believes that the causes for this peculiarity are certain neuropathic changes. I need hardly mention that this hypothesis, of course, throws no light on the subject.

[The inference is drawn from this lucid and comprehensive review of the subject of the etiology of gastric ulcer that when, from any cause, there occurs a devitalization of a portion of tissue in the gastric mucosa, the gastric secretions will digest the same, thus resulting in typical gastric ulcer. What it is that leads to this supposititious tissue defect, keeping in mind the relations of the disease as to the situation of the ulcer, the age and sex of the patient, and the well-known resistance to recovery, is a problem which continues to puzzle clinicians.

In regard to the neurotrophic theory of the affection, above referred to, the exact words of Stockton[1] are as follows: "Unquestionably, the impoverished condition of the blood, leading to lowered resistance of 'the living cells,' and the persistent presence of hyperchlorhydria, must, of necessity, put the tissues to severe strain, but there is wanted yet another factor. The object of this paper is to suggest that, by the influence of some process analogous to herpes, or to idiopathic hæmatoma auris, or to Raynaud's disease, or to herpetic gangrene,—some distinct and persevering nerve-perturbation,—we may best explain the recognized and unaccounted-for feature of the clinical history as to location, age, and sex"; and, it may be added, resistance to healing.

The neurotrophic theory continues to excite interest. One of the last contributions is from R. da la Vedora,[2] in which he records the results of 43 animal experiments, which included excision of the vagus, injection into that nerve of absolute alcohol, and injection of absolute alcohol into the celiac plexus and into the splanchnic. In all experiments on the vagus the results were negative, while following experiments on the celiac plexus there was a decided change in the structure of the gastric mucosa, at the usual site of gastric ulcer, near the pylorus. These changes consisted in erosions and necrosis in 5 out of 12 cases, or 41 per cent. In experiments on the splanchnic identical changes resulted in 9 out of 15 experiments, or 60 per cent. In one case of resection of the celiac plexus the animal died of hametemesis eight days after operation. It is also significant that the gastric juice was much increased in acidity; in other words, there was an accompanying hyperchlorhydria.

Perhaps da la Vedora's failure to succeed with experiments on the vagus was the result of his technic. Yzeran[3] succeeded quite uniformly in producing gastric ulcers in rabbits by making sections in the vagus below the diaphragm. The lesions were generally single, occurred in the pyloric region, and in some instances continued for many months. In 9 of 21 rabbits that were allowed to live for more than two weeks ulcers were demonstrated, and in one animal the ulcer was found over nine months after the vagotomy. Somewhat similar results were obtained by Saitta.[1] Experimental researches of this character must be looked upon as inconclusive so far as they explain the origin of peptic ulcer in man, but at any rate they show that the nervous system, under certain circumstánces, may be competent to excite ulceration of the stomach. There is need of study as to the effect of chlorosis and other affections upon nerve structures in the abdominal cavity.

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[1] *Med. News*, January 14, 1893.

[2] *Boas' Arch. f. Verdauungskrankh.*, vol. viii., No. 3.

[3] *Zeitschr. f. klin. Med.*, 1901, vol. xliii., 81.

Occasionally perforative ulcers occur synchronously in different regions of the body. For instance, Kavetsky[2] describes an autopsy in which, besides a perforative ulcer of the bladder, there was a simple round ulcer in the lesser curvature of the stomach.

In view of the above and many other facts that might be adduced, it does not seem proper to dismiss a hypothesis which appears to be freer from objections, all things considered, than those usually marshalled to explain the occurrence of this mysterious affection.—Ed.]

In conclusion, I must say a few words in regard to an objection that has recently been formulated against the significance of increased hydrochloric acid secretion in the etiology of ulcer. I do this chiefly because I fail to find any opposition to this theory in the different literature references that I have read.

Du Mesnil recently reported the case of a woman of thirty-eight years, in whom a large ulcerated and perforating carcinoma and a small typical funnel-shaped ulcer were found together in the stomach. According to postmortem findings, there was no doubt that the carcinoma was the older lesion, and that the ulcer originated much later. Du Mesnil argues that in this case we can hardly speak of a typical ulcer, for the digestive powers of the gastric juice must have been reduced for a long time before this ulcer developed. From this he concludes that normal, or even hypernormal, acidity of the gastric juice plays no rôle in the causation of gastric ulcer. He goes so far as to say that superacidity is without significance in the formation of an ulcer.

No one will deny that an injury to the gastric mucosa may cause an ulcer in a healthy subject. We need not be surprised occasionally to find a defect of the gastric mucosa in addition to a carcinoma. The only fact that is really surprising is, as we have already mentioned, that typical ulcer of the stomach heals with such difficulty. Hyperacidity need not exist in order that an ulcer be formed, but it must be present in order that an ulcer become chronic. Whether or not the ulcer in this case would have become chronic, no one can say; all that we learn from the postmortem findings is that it was of recent date. But even if it had persisted for a long time, it would not disprove that ulcers, as a rule, are accompanied by hyperacidity; nor would it refute the hypothesis that, in the majority of cases of ulcer, hyperacidity is the most important factor that impedes recovery. [Hydrochloric acid may sometimes be found persistently absent in otherwise typical cases of gastric ulcer, instances of which Ageron has recently contributed[3].—Ed.] There are undoubtedly other causes that render it difficult for the stomach to replace a loss of substance; we may assume, for instance, that a large quantity of lactic acid renders healing of an ulcer difficult. If the ulcer is large and very deep, its margins can no longer come in contact with one another, so that the only way in which an ulcer can finally heal is by the formation of a deep cicatricial defect.

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[1] *Gaz. Degli Ospedeli*, Milan, 1900, vol. xxi., 599. [2] *Prag. Vratch*, 1902, No. 24.

[3] *Münch. med. Wochenschr.*, July 29, 1902.

Pathologic Anatomy.—Round or peptic ulcer is found only in those portions of the digestive apparatus that are exposed to the action of gastric juice—namely, the stomach, the first portions of the duodenum, and the lowest portions of the esophagus.

The most frequent seat of gastric ulcer is the posterior surface of the stomach-wall; next in order of frequency the pyloric portion of the stomach and the lesser curvature. Only 20 per cent. of the ulcers are found on the anterior wall of the stomach. According to Brinton, who collected 220 cases, the distribution of the lesion in different localities of the stomach is as follows: Posterior wall of the stomach, 42 per cent.; lesser curvature, 26 per cent.; pylorus, 15.6 per cent.; anterior wall, 4.9 per cent.; greater curvature, 2.4 per cent.; and cardia, 2 per cent.

The ulcer, therefore, in the great majority of cases is located within a relatively circumscribed area of the stomach—that is, the posterior surface, the lesser curvature, and the pyloric portion. The greater portion of the organ, namely, the fundus, the anterior surface, the greater curvature, and the cardia are *in toto* affected in only one-fifth of the cases.

Number.—As a rule, only one ulcer is found in the stomach; less frequently two or more. One or several cicatrices of old ulcers are quite frequently seen together with more recent ulcers that have not yet healed by cicatrization. Ewald quotes a case of Lange in which so many ulcers were present that he was unable to count them. Brinton, in a total number of 463 cases, found 57 cases with two ulcers, 16 cases with three, 3 cases with four, 2 cases with five, and 4 cases with more than five ulcers.

Form, Appearance, Extent.—Rokitansky described the appearance of gastric ulcers as follows: They look as if they were stamped out with a die; the round, sharp outline of the lesion is usually considered characteristic for gastric ulcer, and hence the common name, "round ulcer of the stomach." The ulcers are occasionally more elongated and oval in outline. As a rule, the round or oval outline corresponds to the capillary distribution of some arterial branch.

The size of the ulcers varies greatly. They may be from 1 to 4 cm. in diameter or much larger. Bouveret, for instance, quotes a case in which the ulcer was larger than half an adult hand. A case that Cruveilhier reported is frequently quoted; here the ulcer was 8 cm. broad and 16 cm. long. The depth of the ulcer also varies: it may extend to the muscularis or even to the serosa. As a rule, however, the different layers of the stomach-wall are not uniformly eroded. This is explained from differences in the distribution of the capillary branches of the artery that is involved.

The loss of substance is usually greater in the mucosa than in the submucosa, and in the latter still greater than in the muscularis. This gives the ulcer a flat, funnel-shaped, or terraced appearance. Ulcers are only funnel-shaped, however, when they are relatively recent; if they are old and extend over large areas, they do not, as a rule, present this appearance, for here the loss of substance is frequently as great or even greater in the deeper layers of the stomach-wall than in the mucous lining. In very large old ulcers the muscularis is frequently pulled up to the mucosa and rolled inward underneath it; the mucosa itself is also rolled inward so that the margins of the ulcer appear like bolsters. Occasionally the ulcer extends through the whole wall of the stomach and leads to the formation of adhesions with neighboring organs—for instance, with the pancreas and the liver. In cases of this kind the ulcer appears as a deep cavity of very irregular shape, with a cicatricial base. If the ulcers are very recent,—so recent that the funnel shape is still distinctly visible,—the axis frequently runs diagonally, and follows the direction of some large vessels of the stomach-wall. The base and the margins of the ulcer are usually smooth, rarely ragged.

Round ulcer of the stomach, provided it is recent and does not extend so deep down into the stomach-walls that the muscularis is destroyed, may heal. This is shown from the frequent appearance of ulcer scars in the stomach. These cicatrices may vary greatly in size; frequently they are so small that they can be found only on careful inspection; in other cases they are very large, stellate in appearance, and permeate the whole stomach-wall. They may be so large that they lead to a contraction of large portions of the stomach-wall. The situation of the scar will often determine whether the stomach assumes an hour-glass form or whether stenosis of the pylorus or the cardia develops.

Hauser, above all, has contributed a great deal to our knowledge of the processes of healing that occur in ulcer of the stomach. According to this author, healing occurs by regenerative proliferation of the connective and glandular tissues that are in immediate proximity to the defect. The cells of the mucosa near the margins of the ulcer chiefly undergo proliferation. Even though very large ulcers do not heal altogether, we will usually find proliferation of gland-cells in the mucosa at the margins of the lesion. Hauser frequently found a large number of tubules in the center of scars that were the result of healed ulcers of the stomach. These tubules were placed vertically to the epithelial surface of the stomach; some of them, however, ran diagonally and others parallel to the mucosa. They were either as broad as ordinary gland-tubules or they were in a state of cystic dilatation. They were not lined by glandular epithelium, but by a species of cylindric epithelium. None of these tubules had an open lumen. Hauser states that these peculiar structures no longer perform a secretory function, but are merely adenoid neoplasms. He adduces this chiefly from the nature of the cells lining the tubules.

As the scar tissue contracts, the proliferation of glands is stopped. If the whole stomach-wall is perforated by the ulcer, the connective tissue in the neighborhood of the stomach—for instance, the mesentery—may grow into the hole and unite with the connective tissue that grows out of the muscularis and serosa of the stomach. In this way the ulcer is frequently healed.

It seems that the muscle-fibers do not regenerate. Near the ulcer the muscle-fibers are separated by connective tissue, and a cicatrix of this kind may frequently be recognized by these strands of connective tissue, even though the hole is completely filled out and all irregularities on the inner surface of the scar have disappeared, owing to the regenerative growth of glandular and connective tissue and the contraction of neighboring portions of the mucosa (Ziegler).

Hauser states that very large and deep ulcers that penetrate through the muscularis and lead to a rolling inward of the mucosa and the muscularis and to adhesions with neighboring organs never completely heal—that is, the loss of substance is never replaced, as the scar tissue that is formed at the base of the ulcer never contracts sufficiently to cause an approximation of the edges of the ulcer. In cases of this kind the gastric juice frequently irritates the tissues to such an extent that a chronic inflammatory proliferation of connective tissue occurs, which may extend downward to the neighboring organs that are adherent to the base of the ulcer. This continuous inflammatory irritation of the ulcer may cause erosion of large or small blood-vessels and thus lead to fatal hemorrhage. At autopsy occluded stumps of blood-vessels, and even open blood-vessel lumina are quite frequently found at the base of such ulcers. If large vessels, like the coronary arteries of the stomach, or the pancreatic, splenic, gastro-epiploic, or gastroduodenal arteries, are eroded, the hemorrhage is usually fatal at once.

If the ulcer penetrates to the serosa before solid adhesions are formed, perforation may occur. This accident happens most frequently when the ulcer is situated in the anterior wall of the stomach, for the reason, chiefly, that this portion of the organ is more freely movable than others, so that solid adhesions do not form as frequently here as in other locations.

If perforation occurs before partial adhesions have formed, acute, general peritonitis that is usually rapidly fatal naturally develops. As a rule, however, death occurs so soon after the perforation that pronounced peritonitic lesions are not observed. In other cases, again, in which adhesions with neighboring organs are first formed, partial peritonitis, usually purulent in character, develops. Sacculated pus foci are formed that are usually situated in the epigastrium or the left hypochondriac region. If perforation occurs in the pyloric region of the stomach, pus may accumulate in the right hypochondriac region. Occasionally pus gathers between the liver and the diaphragm, or, if the perforation occurs on the left side, underneath the left half of the diaphragm. If this happens, the diaphragm is forced upward in this place and may reach even to the fourth or the third rib. The upper wall of such a cavity, which may be filled with pus alone or with pus and gas, is formed by the diaphragm ; the lower wall is formed by the stomach and the liver or other organs, depending on the extent and the localization of the pus focus. We distinguish between pyothorax subphrenicus and pyopneumothorax subphrenicus (Leyden), and speak of the former condition if the pus-sac contains only pus, of the latter if it contains pus and gas. Occasionally particles of food are found in these pus foci ; the pus contained in these sacs is also occasionally fetid. Sometimes subphrenic abscesses of this kind heal spontaneously ; this occurs if the pus perforates the abdominal wall ; it may, however, perforate secondarily into the peritoneal cavity, and in this way lead to fatal purulent peritonitis.

Finally, it may happen that the ulcer perforates into neighboring organs with which partial adhesions had been previously formed. The ulcer, for instance, may perforate into the pancreas. Rokitansky has reported a case in which the posterior wall of the ulcer was formed by the exposed pancreas ; here a voluminous piece of pancreas had become separated at the base of the ulcer and had dropped into the stomach. Cases have also been observed in which the ulcer perforated into the liver or the gall-bladder. Splenic tissue rarely forms the basis of an ulcer ; this is due to the fact that that portion of the stomach that is in close proximity to the spleen is rarely affected by ulcerative processes. A few cases have been reported in which adhesions formed with loops of intestine, and in which, subsequently, perforation of the ulcer into the intestine occurred. Perforations into the pleural cavity, into the pericardium, and even into the heart, have been noted. Guttmann, Matthieu, and Moizard have reported cases of pneumopericardium as the result of perforating ulcer of the stomach.

Chronic inflammatory processes in the neighborhood of the ulcer are very frequently found on postmortem examinations ; for instance, perigastritis, partial peritonitis, adhesions with loops of intestine, with the pancreas, the liver, the spleen, and the transverse colon. The case reported by Korach is isolated. This author saw general emphysema develop after perforation of an ulcer. The case of Jürgensen is still more peculiar, for he found air in the blood after perforation of an ulcer. A case reported by Kolaczek is a rarity ; he reported a diverticulum of the stomach that had developed from a gastric ulcer.

The macroscopic appearance of round ulcer is, as a rule, so characteristic that it can hardly be confounded with any other form of ulcer, particularly the infrequent forms of tuberculous or syphilitic ulcer of the stomach. The former are found only if there is tuberculosis of other organs, are irregular in appearance and more irregular in outline, and frequently have a cheesy base, covered with tuberculous nodules. The serosa of the stomach also is covered occasionally by miliary nodules. Tuberculous ulcers in the majority of cases are multiple.

It is very doubtful whether syphilitic ulcers ever occur in the stomach.

[As to the possibility of the occurrence of syphilitic ulcer of the stomach : this, although rare, is believed by most observers to be an actual occurrence; for instance, Schreib[1] describes the case of a man who, after several weeks' illness, died suddenly of hematemesis. At autopsy perforation of the stomach was found at the base of a funnel-shaped ulcer; the surrounding tissues showed this to be the center of a gumma. There were similar areas found in the lung and several parts of the intestine. A similar case was reported by E. Fraenkel, and Dieulafoy[2] states that various forms of syphilitic lesions of the stomach, often leading to ulceration, are not at all infrequent.—Ed.]

Simple follicular ulcers are interesting only from an anatomic point of view; their only clinical significance is that they may occasionally lead to the formation of genuine ulcers.

[A case of gastric ulcer occasioned by the diphtheria bacillus, with autopsy, is reported by Stokes in the *Johns Hopkins Bulletin* for July, 1901.—Ed.]

Symptoms.—Ulcer of the stomach is a disease that can frequently be diagnosed with great certainty from the history of the case. In many instances, however, prolonged observation alone can lead to a correct diagnosis, and in still other cases the disease may run an altogether latent course. Cicatrices of round gastric ulcers are so frequently found postmortem that we must conclude that ulcers frequently remain clinically unrecognized, as in many of these cases no symptoms of stomach trouble developed during life. In other cases, again, the patients may be perfectly well and show no symptoms whatever of ulcer, then suddenly develop hematemesis or symptoms of perforation. I quote cases of this kind from my practice:

Two years ago a gentleman of forty-five years, who resided out of town, came to consult me. I had treated this patient a year before for stomach trouble, his only symptoms at that time being a feeling of slight pressure after eating, and belching. Objectively, neither pain on pressure nor a tumor, nor ectasy, nor any other anomaly could be discovered. Diagnostic analysis of the stomach-contents revealed hyperaciditas hydrochlorica. The patient was placed on a course of Carlsbad waters and his diet regulated. He returned to me afterward because some of the old symptoms had reappeared, though only to a slight degree. The appetite was excellent, the general nutrition very good. As the patient was coming to the clinic the second time and was just entering the door he suddenly felt dizzy; he dragged himself to the anteroom and fell down in a faint. As I happened to be present I could observe everything that occurred myself. He turned very pale, the pulse was small and accelerated. He soon recovered and complained of nothing but a feeling of faintness and weakness. I assumed at that time that a hemorrhage had occurred, probably from a gastric ulcer. As a matter of fact, the stools that were evacuated showed an abundant admixture of blood. The subsequent course of the disease, which I will not describe in this place, corroborated our suspicion of the existence of a gastric ulcer.

Another example may be quoted. The patient in this instance was an architect, a very robust and healthy-looking man. One day, as he was leaving his house right after dinner, he suddenly collapsed on the street in front of his house. Before he fainted he uttered a loud cry. As I lived in the neighborhood I was sent for. On examination I found the abdomen very much distended, under high tension, and painful. The liver dulness had disappeared, and there was a uniform tympanitic sound all over the abdomen; in short, there were all the symptoms of a free perforation into the abdominal cavity. Peritonitis developed rapidly and the patient died within thirty-six hours. The autopsy revealed the presence of a perforated gastric ulcer with escape of gas and stomach-contents into the abdominal cavity. It was established in this case that the patient had never been in the care of a physician and had never complained of any stomach symptoms.

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[1] *Prager med. Wochenschr.*, Nos. 45, 46.

[2] *Bulletin médical*, 1898, No. 40.

Such latent forms are undoubtedly rare; as a rule, a careful history of the case will reveal that mild dyspeptic symptoms were complained of some time before the hemorrhage or the perforation occurred. Frequently these preliminary symptoms are so insignificant that they do not lead us to suspect the existence of an ulcer; we should certainly err, however, in excluding the possibility of ulcer in all cases where pronounced symptoms of this condition are absent. In many cases we shall, at least, be able to suspect ulcer or to make a probable diagnosis, and shall have to make our final decision dependent on the course of the disease and the result of the treatment instituted.

General Picture.—We will first discuss the general picture that is seen in the majority of cases of ulcer. As a rule, the symptoms develop slowly, not suddenly. In the beginning there will merely be a feeling of pressure after eating, particularly after eating indigestible food; this distress gradually increases, and finally develops into violent attacks of pain, vomiting, and similar symptoms. The patients, above all, seem to complain of periodically recurring attacks of pain immediately after eating; usually these attacks occur relatively soon after eating—that is, from one-half to one or two hours afterward. In rare instances they appear while the patients are eating or immediately afterward. The duration of these attacks varies, but they usually persist throughout the whole time of digestion, and usually stop as soon as the ingesta are propelled from the stomach into the intestine. In other cases vomiting occurs. The vomit tastes very acid and consists of a more or less abundant quantity of acid food. In rare instances patients vomit during the night or early in the morning. Occasionally no food is raised when the patients vomit in the morning, but only a cloudy, very acid fluid that contains a few flakes of solid material. This is seen particularly in some cases of continuous secretion of gastric juice, a disease that, as we have already seen, is quite frequently associated with ulcer. Sometimes this late vomiting is due to the coexistence of ectasy of the stomach. In pure, uncomplicated ulcer, however, vomiting usually occurs very soon after eating—that is, within one to three hours afterward, rarely later. As soon as vomiting occurs the pain usually stops, or it may persist for a little while longer; in the latter case the pain is not so severe. The pain is usually limited to the gastric region, but may also radiate to the back or still further. Objective examination, as a rule, reveals a painful pressure-point in a small circumscribed area of the epigastric region. The appetite varies; in some cases it is slight; in others the patients have a desire for food, but do not dare to eat solid food or large quantities of food because they fear the attacks of pain that they know will follow. This symptom-complex may remain unchanged for some time. Suddenly a violent gastric hemorrhage may occur that may be repeated several times and that always exhausts the patient. In other cases vomiting of blood does not occur, but the patients notice that, instead of looking as well as they had, they seem to be growing paler, to feel weak and relaxed, and that the pulse grows very small and accelerated. An experienced practitioner, who should consider the diagnosis of ulcer from the symptoms that we have described, will always think of hemorrhage as soon as anemic symptoms of this kind suddenly appear, and will instruct the patients to save their stools. Even though no vomiting of blood occurs, there may have been a gastric hemorrhage, and the stools of these cases will be very black and contain blood. As soon as this occurs, the disease ceases to be a mild affection; and as soon as hemorrhage occurs, the symptoms usually become more severe.

Whereas the daily occurrence of attacks of pain, frequent vomiting, and deficient nourishment weaken the patient only to a slight degree, an abundant hemorrhage leads to very great exhaustion. However severe the anemia may be, the patient, as a rule, does not present a cachectic appearance until further complications occur.

Another important symptom of this condition is the following: If the stomach-contents is analyzed after a test-breakfast or a test-meal, or if the vomit is examined, hyperaciditas hydrochlorica may be found. I need hardly mention that aspiration of stomach-contents for diagnostic purposes should not be performed immediately after a gastric hemorrhage.

Another feature that may be briefly mentioned, and that is occasionally seen together with the other symptoms, is more or less obstinate constipation.

If all these symptoms are present, the diagnosis of ulcer is, of course, not difficult. No single symptom can be regarded as characteristic, but the whole symptom-complex, particularly if it occurs in youthful patients, hardly allows us to diagnose anything else than ulcer. This whole symptom-complex, however, is by no means present in all cases: this, that, or the other symptom may be absent; there may be only mild dyspeptic disturbances, similar to those we see in many other conditions. Variations in the syndrome may even be seen in the same case during the course of the disease; temporary remissions and exacerbations may occur, or the case may be protracted for months or a year, or even longer. In other cases, again, all the symptoms enumerated above may persist for a long time and then gradually decrease in severity; vomiting and the attacks of pain may stop, and the general strength and the appearance of the patient may improve; suddenly, however, even though no particular indiscretion in diet, etc., has been committed, the symptoms exacerbate, hematemesis occurs, and this at a time when every one believed that the ulcer was partially or altogether healed. In this way the course of the disease may be protracted for a long time. As long as the ulcer is not completely healed it must be remembered that there is always danger of perforation. In some instances we may see a gradual disappearance of the ulcer symptoms proper, but at the same time the stomach will be more or less dilated, there may be adhesions of neighboring organs, or other sequelæ.

Round ulcer of the stomach may heal completely without leaving any sequelæ. This occurs quite frequently if relatively recent forms of ulcers are correctly treated; ulcers of this kind frequently heal in a short time. If the ulcer covers a large area and is very deep, or if it persists for a long time or is complicated with hypersecretion, the chances of recovery are less favorable.

This may represent a brief description of the symptom-complex of ulcer that is seen in many cases. We have already called attention to the fact that deviations from this syndrome are frequently seen. Following this short description of the general picture we will proceed to analyze single symptoms.

The Pain.—The most prominent symptom of ulcer is pain. The pain of ulcer has several important characteristics: it appears in paroxysms, it is strictly localized, it occurs at the time of digestion, and is influenced by the character of the food. The location of the paroxysmal pain usually corresponds to the place that is most sensitive to pressure. The pain from ulcer usually possesses all these peculiarities; there are, however, a number of exceptions, all of which are due to some particular cause. Occasionally the pain is continuous and does not occur in paroxysms only at the time of digestion; if the pain is continuous, this indicates some complication and does not speak for simple ulcer. Simple ulcer of the stomach produces pain only when the organ is irritated, and the most common irritant is the food. When the stomach is empty and is not irritated in any way, the ulcer is rarely painful; occasionally the patients complain of a dull, disagreeable feeling in the gastric region; as soon, therefore, as continuous pain of some severity appears, we must think of some complication. If such continuous attacks of pain appear acutely in the course of an ulcer of the stomach, we must always think of circumscribed peritonitic inflammation. Sometimes peritonitic adhesions cause continuous pain, or it may be caused by old ulcers that penetrate through the wall of the stomach and extend to some neighboring organ. More frequently the pain that is caused by adhesions is experienced only when the patient occupies certain positions or if the stomach is more distended than usual. In many cases, after the symptoms of ulcer itself have disappeared, pain is felt in certain portions of the abdomen only when the stomach is filled and not in the interval. I remember, for instance, the case of a student who was at one time afflicted with ulcer, and in whom these peculiar symptoms developed after the symptoms of ulcer proper had disappeared; as soon as he ate a large quantity of food while he was sitting up violent drawing pains were felt in a circumscribed area, but this pain stopped at once as soon as he occupied a semirecumbent position. At all other times the patient was free from pain.

But even if no further complications are present, the pain following the ingestion of food may vary in intensity according to the position that the patient occupies. Many cases of ulcer are greatly relieved if they lie horizontally in bed—even those cases that suffer from very violent ulcer pain. Some patients suffer less when they are sitting up;

others when they are lying on their back or on their side. Some investigators are inclined to explain this from the position of the ulcer; they argue that the ingesta come in contact with the ulcer and irritate it, etc., more in one position than in another, and that the position in which the patient is most comfortable will depend altogether on the seat of the ulcer. We have already stated, however, that, as a rule, the attacks of pain coincide with the ingestion of food; as soon as food enters the stomach the secretion of gastric juice is stimulated and, since in ulcer an abnormally large secretion of hydrochloric acid usually occurs, the ulcer and the sensory nerves that are at its base are irritated either by the acid directly or mechanically by the peristaltic movements of the stomach. The paroxysms of pain caused by these agencies do not, as a rule, appear immediately after the meal, but generally some little time afterward—from one-half to one hour or even later. Their duration also varies. It is undoubtedly correct to assume that the increased secretion of acid at this time is concerned in the production of the paroxysm of pain; the paroxysms soon reach their height and remain severe for a shorter or a longer time. Generally vomiting occurs and stops the cardialgic pain, so that the patients merely complain of a dull feeling of soreness and discomfort. The patients usually themselves state that the attacks of pain are directly connected with eating, and that they appear at the height of digestion. This symptom is very important, particularly for the differential diagnosis between carcinoma and ulcer. Cardialgia is also occasionally seen in simple hyperacidity without ulcer, but here the attacks are not quite so severe; they are, however, also dependent on the ingestion of food, and are most severe at the time of digestion. The pain in these cases never occurs as regularly as in ulcer. [As a rule, the pain in ulcer appears earlier than in cases of uncomplicated hyperchlorhydria.—Ed.]

In carcinoma this is different. Gerhardt is undoubtedly correct when he says that those patients who are afraid to eat because they fear the pain are rarely cases of carcinoma, but usually cases of ulcer with cardialgia. The opposite may occasionally be seen—namely, that the ingestion of certain articles of food seems to stop the attack of pain for the time being. This is quite frequently seen in patients who appear to be suffering from ulcer. This symptom, however, primarily has nothing to do with the ulcer *per se;* it is dependent on the production of acid; as soon as this becomes combined with the food, the pain stops for a time. The symptom is particularly frequent in cases of continuous secretion of gastric juice. The latter condition is often seen together with ulcer, or, better, cases of hypersecretion are frequently afflicted with ulcer. In these patients attacks of pain occur not only at the time of digestion, as in uncomplicated ulcer, but also at a time when the stomach is empty, or at least should be empty—that is, most frequently at night, occasionally toward evening, though the patient may have taken no food since the midday meal. Occasionally the pain is felt before meals. If these patients are instructed to eat an egg or something else, the pain usually stops, though, as a rule, only for a short time.

As a matter of fact, it is easy to determine that these attacks of pain have nothing to do with the ingestion of food. If the stomach-contents is aspirated at the height of an attack of pain, the organ will be found empty—that is, it will contain no remnants of food; it is not absolutely empty, however, for a yellowish, cloudy fluid is poured out through the sound, which varies in quantity, and on examination will be found to be gastric juice. Normally the stomach is empty after fasting for a long time. If acid gastric juice is secreted when the stomach contains no food, peristaltic movements are excited and at the same time the surface of the ulcer is irritated; if, now, these patients eat something, the hydrochloric acid is combined with the proteids and the pain stops. Indirectly, therefore, the ulcer is concerned in the production of this pain, but the primary causative factor is the abundant quantity of gastric juice that is secreted when the stomach is empty.

In cases of ulcer of this character, therefore, in which there is a continuous secretion of gastric juice, two kinds of paroxysms of pain may be observed: one occurs at the height of digestion, as in simple, uncomplicated ulcer; the other in the intervals between digestion, particularly at night.

The character of these attacks of pain is described differently: some patients state that it is burning, others stabbing, still others, spasmodic in character. Pain may be felt in a circumscribed area or may extend over a large portion of the abdomen—may reach even to the thorax, to the arms, and to the spinal column. External pressure usually exacerbates the pain. This point may be utilized in the differential diagnosis between ulcer and neuroses of the stomach, for in the latter condition the pain is usually relieved by pressure. The fact that ulcer pain is made worse by pressure explains the fact that patients with ulcer can wear no tight clothing—corsets, etc. The slightest pressure frequently causes great pain.

There is a certain diversity of opinion in regard to the question whether or not the cicatrix of an ulcer may cause pain. Gerhardt is of the opinion that the continuous pain, and the pain that occurs when the patient occupies certain positions, may be due to adhesions between the ulcer or the cicatrix from an ulcer with other organs. Leube states that cicatrices are a very uncommon source of pain; and warns against diagnosing cicatrization if the pain and certain dyspeptic symptoms persists in an ulcer case after the patient has undergone a Carlsbad cure or a rest-cure.

We must always remember that cardialgic ulcer pain appears in paroxysms, and is usually very severe. The occurrence of these attacks is dependent on the ingestion of food, and also to a certain extent on the quality of the food. The pain is relieved by a non-irritating diet and by rest. The coarser the food, the more does it irritate the stomach both mechanically and chemically, and the more violent is the pain. What applies to ulcer does not apply to cicatrization *per se*, for this, as a rule, causes no pain; it can lead to attacks of pain only if adhesions form; even adhesions can be made responsible for paroxysms of pain, or cardialgia, only if they form in such a manner that certain portions of the stomach-wall are pulled and distorted whenever the organ becomes distended and filled with food. I agree altogether with Leube when he says that cicatrices from gastric ulcer cause pain only in very rare cases. The pain caused by adhesions and cicatrices appears only when the stomach is filled to a certain degree. Attacks of pain are not dependent on the quality of the food, as in ulcer, but on the quantity. In many of these patients the pain appears only when they occupy certain positions, and is rarely continuous. The character of the pain is also different; the patients complain of a feeling of tension or traction in a circumscribed spot, whereas patients with ulcer complain of spasmodic pain. In ulcer cases there are frequently painful pressure-points, whereas adhesions or cicatrices are not sensitive to pressure, or at most painful only to a slight degree. All these points, of course, are not absolutely reliable criteria, but, as a rule, a careful examination of the patient, a study of the general picture of the disease and of its course, will enable us to differentiate between pain that is caused by cicatrices or adhesions and pain that is caused by ulcer. In doubtful cases the decision will have to be rendered through the results obtained from treatment directed against the ulcer. Inflation of the stomach in cases where adhesions are suspected may occasionally aid the diagnosis.

I have already mentioned that the quality of the food exercises a great influence on the production of pain in ulcer. We need hardly elaborate on this point, for we see this in a more or less pronounced degree in every case of ulcer. One of the most important factors in the treatment of these cases is to administer a non-irritating diet—that is, a diet that is neither mechanically, nor chemically, nor thermically irritating to the stomach. If treatment of this kind is instituted, the pain usually disappears in a short time, only to reappear, however, as soon as indigestible food is eaten. If we put the stomach completely at rest; if we feed the patient *per rectum* for several days; the pain, as a rule, stops altogether. Milk or other liquids are generally well borne, whereas solid food usually causes pain. The intensity of the pain varies according to the amount of gastric juice that is secreted after the ingestion of different articles of food. It is impossible to arrange a scale of intensity of pain for different articles of food, for certain individual factors are concerned in the production of this pain. Many subjects can eat one article of diet with impunity that may cause heartburn, acid eructations, or even cardialgia in another subject. Some patients are sensitive to hot drinks, others to very cold ones; in other words, the stomach in these cases appears to react with particular energy to thermic irritants. Other factors may influence the intensity of the pain; in women, for instance, the distress is frequently greater at the time of menstruation than at other times.

The location of the pain varies in different cases, but is always in the same location in the same individual. As a rule, the pain is not merely felt in the location of the ulcer, but extends beyond; the painful pressure-points, however, are always strictly circumscribed and located where the ulcer is. The cardialgic pain, which is dependent on eating, may or may not radiate; everything will depend on the intensity of the paroxysms and the seat of the ulcer. If the attack is very severe, the pain may radiate over the whole gastric region or even beyond; as a rule, it is felt in the epigastric region; in other cases it is felt in the back; it may radiate into the hypochondriac region of either side, and toward the shoulder-blades; in fact, it may radiate into the brachial plexus and the fibers of the pneumogastric supplying the lungs (Traube, Rosenheim). As a rule, however, that portion of the stomach that is painful to pressure corresponds to the location of the most severe pain in the cardialgic attacks.

Some authors have attempted to draw conclusions in regard to the seat of the ulcer from the time that elapses between the ingestion of food and the onset of the paroxysm of pain. They claim that the ulcer is always localized in the pyloric region if the pain appears late; that it is localized in the posterior wall if the pain appears early; and that it is localized in the cardia if the pain occurs as soon as food enters the stomach. I do not think that much importance can be attached to these considerations. It makes very little difference where the ulcer is located, for pain will appear as soon as the peristalsis of the stomach is excited by the ingestion of food and as soon as the gastric juice comes in contact with the surface of the ulcer. I do not think that we can draw any definite conclusions in regard to the seat of the ulcer from the time that elapses between the ingestion of food and the beginning of the pain. If pain occurs simultaneously with swallowing, this may possibly be considered an argument in favor of an ulcer in the cardiac region of the stomach.

The position that the patients occupy during a cardialgic attack varies. The majority of patients occupy a position in which the abdominal walls are relaxed—that is, they bend over forward; others again occupy a horizontal position; still others lie on their stomach or on their side. The application of heat, as a rule, relieves the pain.

Pain on Pressure.—An objective examination of ulcer cases, as a rule, shows that certain portions of the gastric region are painful to pressure, so that this symptom is noticed in addition to the spontaneous paroxysm of pain. We distinguish between painful pressure-points in the epigastric region and pressure-points in the dorsal region. The epigastric pressure-point is usually situated in the median line or to the left of it; less frequently, to the right. The point of greatest pain is usually situated immediately below the ensiform process. If the stomach is dislocated downward, the pressure-point is situated lower down. Brinton has written a minute description of this epigastric pain that is elicited on pressure, and has called attention to the fact that in women the painful point is very near the umbilicus, owing to the fact that corsets press the stomach downward.

The region that is sensitive to pressure varies in extent. As a rule, pain is elicited over a relatively small area; occasionally, however, the pain radiates from this point into the surrounding parts. Sticker[1] has recently shown that in hysteric hyperesthesia sensitiveness to pressure is felt over an area that corresponds to the outline of the stomach, even in those cases in which the organ is artificially enlarged. In contradistinction, therefore, to the pain of ulcer that is strictly localized within a small area, the pain in hysteric hyperesthesia corresponds exactly to the size, the outline, and the position of the whole stomach.

In addition to epigastric pain we occasionally encounter dorsal pain on pressure. Cruveilhier was the first to describe this pain. The painful pressure-point is usually situated at the height of the seventh to the twelfth thoracic vertebra; in general, a little to the left. The pressure-point, as a rule, is small—about 3 to 4 cm. in diameter. According to Boas, this pressure-point can be found in about one-third of all cases of ulcer; it is rarely found alone; according to Bouveret, both painful pressure-points, the epigastric and the dorsal one, are present together in the majority of cases.

Boas has devised a special instrument, called an algesimeter, for the purpose of measuring the pain on pressure. In round ulcer of the stomach an examination with this instrument frequently shows that a weight of from $\frac{1}{2}$ to 1 kilogram causes pain, whereas in chronic gastritis 4 to 6 kilograms can be borne without pain. Boas states that values over 4 kilograms speak against round ulcer of the stomach. This pain on pressure is undoubtedly of diagnostic value; whereas in cardialgia of a purely nervous character pressure exercised in the epigastric region frequently modifies the pain, in gastric ulcer relatively slight pressure frequently causes very severe pain.

The attempt has also been made to determine the seat of the ulcer from the location of the painful pressure-point. The results observed so far, however, are uncertain. If pain can be elicited by pressure in the dorsal region, but not in the epigastric region, we may think of ulcer of the posterior wall of the stomach.

Vomiting.—Vomiting is less constantly seen than cardialgic attacks. As a rule, the two symptoms appear together, vomiting usually occurring at the height of an attack, less frequently immediately after eating. At the same time vomiting is not seen in every case of cardialgia. Many patients with ulcer suffer from nausea and belching, but only in exceptional cases from vomiting, even though the attacks of cardialgia may be quite severe. On the other hand, we frequently see cases that vomit regularly one or several hours after each meal, and do this for a prolonged period of time. We see still other cases that vomit only at irregular intervals.

While vomiting is a very important symptom of ulcer, it is by no means a constant one. From a diagnostic point of view the paroxysms of pain are more important than attacks of vomiting; although the former by no means demonstrate the presence of an ulcer, there are few other diseases of the stomach in which attacks of this kind occur with such regularity. We can say the same of vomiting, for while this symptom occurs in a great variety of affections of the stomach, it has certain peculiarities in ulcer; as a rule, it occurs at the height of the paroxysm of pain—within two or at most three hours after a meal. In many other diseases of the stomach,—for instance, gastrectasy, hypersecretion, etc.,—vomiting, as a rule, occurs much later and at irregular intervals. In ulcer, in addition, vomiting seems to be directly dependent on the quality of the food, just like the attacks of pain. We have already mentioned that the attacks of cardialgia disappear rapidly if the patient is fed on a liquid diet, particularly a milk diet; the same applies to vomiting. On the other hand, coarse and indigestible food frequently leads to cardialgic attacks and at the same time causes vomiting. In general it may be said, therefore, that there is a certain parallelism between the severity of the cardialgic attack and the vomiting. There are, however, numerous exceptions to this rule. It appears that some patients vomit easily and others with difficulty. If vomiting occurs in ulcer cases during the cardialgic attack, the paroxysm of pain usually stops, for the evacuation of the ingesta usually removes the cause of the attack.

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[1] *Zeitschr. f. klin. Med.*, vol. xxx., Nos. 1, 2.

The appearance of the vomit may vary; much will depend, on the one hand, on the character of the food, and, on the other, on the time at which vomiting occurs. The longer the time between the ingestion of food and vomiting, the more completely digested will the vomited material be. As a rule, ulcer patients digest very rapidly provided there are no complications; the digestion of albumin in particular is very good, because the production of hydrochloric acid is, as a rule, increased; consequently the material that is vomited in these cases is quite characteristic, namely, very fine; it possesses a distinctly acid odor, and the patients complain of an acid taste and state that the teeth feel dull after vomiting. Chemical examination usually reveals high acidity; that is caused by high values for hydrochloric acid, not for fermentative acids. If vomiting occurs very soon after a meal, the value for hydrochloric acid will, of course, not be so high. All this is different in cases that are complicated in some way. We see, for instance, continuous secretion of gastric juice together with ulcer, and this condition is frequently, though not always, complicated by ectasy and atony of the stomach; or, again, ectasy may be caused by a stenosis of the pylorus. If there are hypersecretion and ectasy, vomiting, as a rule, occurs some time after eating, and in many instances late at night or early in the morning. Sometimes the vomit shows no particles of food, but consists of a cloudy fluid containing much hydrochloric acid; in other instances the patients vomit coarse particles of food that are mixed with abundant quantities of mucus. This is seen particularly in cases of old ulcers with secondary catarrh of the stomach.

Vomiting of Blood.—Many consider the vomiting of blood a symptom that is particularly characteristic for ulcer of the stomach. This symptom, however, is absent in very many cases of ulcer; and, on the other hand, it may be due to a variety of other causes. It occurs in different forms of erosion of the gastric mucosa, in tuberculous and carcinomatous ulcers, also in a number of circulatory disturbances of the stomach-wall, particularly with cirrhosis of the liver. I saw a fatal hematemesis in one of my colleagues who was suffering from incipient cirrhosis of the liver. The patient suddenly had a violent hematemesis while he was feeling perfectly well; he recovered from the first attack, but six weeks later another violent hemorrhage occurred to which the patient succumbed. No trace of round ulcer of the stomach was found. Hematemesis may occur even though there is no true hemorrhage into the stomach. I have seen a number of cases in which a ruptured varix of the esophagus caused the bleeding.

Even in those cases where we know that there is an ulcer of the stomach and in which hematemesis occurs, we cannot positively say that the blood comes from the blood-vessels of the stomach-wall. Old gastric ulcers frequently extend to neighboring organs; as gastric ulcers are most frequently found in the pyloric portion, in the posterior wall, and in the lesser curvature of the stomach, the ulcer is liable to extend to the pancreas, the left lobe of the liver, and less frequently to the spleen. Arteries belonging to these organs may, therefore, become eroded by the ulcer and cause hemorrhage.

The statements of different authors vary in regard to the frequency of hematemesis in ulcer. According to Brinton, gastric hemorrhage is found in 28 per cent. of the cases. Witte, in Copenhagen, saw vomiting of blood 100 times among 339 cases. Gerhardt found it in 47 per cent. of his cases. L. Müller, 35 times among 120 cases—that is, in not quite one-third of the cases. Lebert saw hematemesis in all but 22 of 104 cases of gastric ulcer. Among 33 cases of gastric ulcer that were examined postmortem he found a history of hematemesis only 10 times. We must never forget, however, that hemorrhage, particularly if it is slight, may easily be overlooked, and that small quantities of blood may be present in the stools and remain unrecognized. This is particularly liable to happen if the general health of the patient is not impaired. There can be no doubt that if these small hemorrhages were always discovered and were included in statistical calculations, the frequency of hematemesis in ulcer would be found to be greater. Bleeding is usually due to erosion of a blood-vessel by the ulcer. Hematemesis is favored by physical exertion, by emotional disturbances, by serious acute diseases,—for instance, the hemorrhagic diathesis in typhoid (Gerhardt),—and, in women, by menstruation. The violence of the hemorrhage, the amount of blood lost, the rapidity with which the blood is shed, all depend on the size of the eroded vessel.

Very severe hemorrhages may lead to instant death before any blood is vomited or passed in the stools. These cases are always due to erosion of a large artery; the patient suddenly faints, turns pale, falls over, performs a few spasmodic movements, and dies within a few minutes with all the symptoms of internal hemorrhage. If the body is opened, the stomach in these cases will be found filled with blood. Generally speaking, such violent and immediately fatal hemorrhages are rare. Cases in which there is violent hemorrhage that does not immediately lead to death are more frequent; here, too, the occurrence of hemorrhage is recognized by symptoms of severe anemia that appear suddenly; in other words, by the well-known symptoms of internal hemorrhage. The patients turn exceedingly pale, the hands and feet become ice cold, the sensorium becomes benumbed, or the patients complain of violent headache and vertigo. The pulse in these cases is very small and greatly accelerated. If the patient does not lose consciousness, he complains of a feeling of fulness and warmth in the epigastrium. Nausea and a tendency to vomiting soon supervene, and in a short time an abundant quantity of dark, partially fluid, partially coagulated blood is vomited. As a rule, the masses of blood that are vomited, even though they are raised shortly after the hemorrhage, are brown-black in color, owing to the fact that the hemoglobin is converted into hematin by the action of the hydrochloric acid. This rapid conversion of hemoglobin into hematin is favored by the hyperacidity that usually exists in these cases. Jaworski and Korczynski claim to have noticed that hyperacidity may even be increased immediately before and after the vomiting of blood.

In very abundant hemorrhages a part of the blood always leaves the body by way of the intestine; the stools evacuated the following day usually show a uniform brown-black color and their surface is shiny. This peculiarity aids us in differentiating hemorrhage occurring in the stomach from hemorrhage occurring in the large intestine, for in the latter case the admixture of blood and feces is not so intimate—the blood is merely deposited on the fecal masses and is always lighter in color than in gastric hemorrhage.

Occasionally in severe gastric hemorrhage no blood is vomited, but all of it enters the intestine. Here, too, sudden anemia will lead us to suspect hemorrhage. It appears that the blood passes into the intestine particularly in those cases in which hemorrhage occurs gradually and slowly, whereas in those cases in which the stomach is suddenly distended by blood, nausea and vomiting occur with greater frequency.

If the blood remains in the stomach for some time, and if it is vomited later together with gastric contents, very few unchanged red blood-corpuscles will be found, sometimes none at all; instead, large and small brownish masses of pigment will be seen; the vomit, in other words, has a peculiar coffee-ground appearance. This is so typical that it cannot be confounded with other discolorations of the vomit that might be caused by red wine, iron, coffee, etc. In doubtful cases it is impossible to arrive at any decision from microscopic examination. This is due to the fact that it is manifestly impossible to determine the presence of red blood-corpuscles by microscopic examination after they have been destroyed by digestion. Teichmann's hemin test cannot be very well employed with stomach-contents.[1] The spectroscopic method is more reliable, but it is difficult and can rarely be employed in practice. The best method is the one described by Weber,[2] for it enables us to demonstrate the presence of blood in the vomit, in the feces, in the urine, or in the sputum.

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[1] Compare page 145.

[2] *Berlin. klin. Wochenschr.*, 1893, No. 19.

The test is performed as follows: A large portion of the vomit is macerated with water, to which is added one-third volume of glacial acetic acid. The mixture is shaken with ether. After the acid ethereal extract has become clear, a few cubic centimeters are poured off and mixed with ten drops of a tincture of guaiac and twenty to thirty drops of turpentine. If blood is present, the mixture will turn bluish violet; if blood is absent, it will turn reddish brown with a slight tinge of green. The reaction is still more distinct if water is added and the blue pigment is extracted with chloroform.

Hemorrhage such as we have described is apt to recur after a few days, notwithstanding the greatest care. I observed a patient in my clinic some time ago who entered the hospital four days after an attack of hematemesis. On the day on which he entered the hospital a second attack occurred. The patient was not allowed to take any nourishment by mouth, and was fed exclusively by nutritive enemata. He was forced to remain in an absolutely horizontal position, and a small ice-bag was kept over the gastric region continuously. Notwithstanding all these measures hemorrhages recurred several times.

There are finally a number of cases in which slight hemorrhages occur. Such mild attacks of hematemesis may easily be overlooked, even though bloody masses are vomited or blood is passed in the stools. Only careful inspection will enable us to discover those brownish masses that lead us to suspect the presence of blood in the vomit or the stools. In doubtful cases Weber's test, which we have described above, may be performed with advantage.

I have already mentioned that it may be difficult to demonstrate that the blood really comes from the stomach. If blood from the gums, the pharynx, or the esophagus enters the stomach, remains there for some time, and is later evacuated by vomiting, it cannot be distinguished from blood that comes from the stomach-walls. The only way in which to determine the origin of the blood is to elicit a careful history of the case, to examine the buccal cavity carefully, the pharynx and contiguous parts, and to determine by every means at our disposal that bleeding occurred from some other portion of the body than the stomach. The differential diagnosis between hemoptysis and hematemesis may be quite difficult in many cases. We refer to the section on Differential Diagnosis for the details.

But even if we can decide with certainty that the blood comes from the stomach, we do not know that it necessarily comes from an ulcer. Hemorrhages in the stomach may be due to a variety of other lesions; the only way in which to determine in any given case whether or not the bleeding is due to an ulcer, is to study the whole symptom-complex. For the details I again refer to the section on Diagnosis. I will merely mention here that profuse hemorrhages may occasionally be due to small capillary erosions of the gastric mucosa. A. Fränkel has recently reported such a case.

Dyspeptic Symptoms.—If we interpret "dyspepsia" to signify a disturbance of digestion, to indicate that digestion is rendered more difficult or is retarded, we can hardly speak of dyspeptic symptoms in many cases of ulcer; for in ulcer, as we will see when discussing the results of analysis of the stomach-contents, digestion is, as a rule, good, even accelerated, excepting in those cases in which there is, at the same time, a severe catarrh of the mucosa, ectasy, or some other complication. The tongue, as a rule, is clean, red, moist, and rarely coated. The thirst is occasionally increased, but more frequently not; increased thirst is seen particularly in those cases in which there is at the same time hypersecretion. The appetite varies; many patients have a craving for food, but are afraid to eat more than a certain quantity because they know that pain will occur. In many instances we find that the appetite is greatly increased, or there may even be bulimia. In rare instances the appetite is completely lost for a prolonged period of time; this occurs particularly in old cases of ulcer that are complicated with other lesions of the stomach. Ulcer patients rarely complain of acid belching or heartburn. If the latter is present, the patients complain of a disagreeable burning sensation that seems to rise upward from the stomach and is felt either behind the sternum or between the shoulder-blades.

The stools are usually normal, occasionally a little sluggish, and in rare cases diarrheic. The sluggishness of the intestine may be due to a variety of factors. Leube in his day believed that this was due to an inability of the stomach to propel the chyme into the intestine, to insufficient impregnation of the food with gastric juice, and to an inhibition of the normal movements of the stomach. Many authors still harbor this belief, but it is not correct; for in pure, uncomplicated ulcer the secretory and the motor powers of the stomach are not only not reduced, but, on the contrary, increased. This constipation, which, by the way, is neither a regular nor a constant symptom, can hardly be attributed to any one uniform cause. In many cases there may be chlorotic or anemic conditions that cause constipation; in others the character of the food may be held responsible; in others the lack of sufficient exercise. Leube is correct when he attaches some significance to the fact that very little stomach-contents is propelled into the duodenum in those cases in which there is persistent vomiting. Bouveret argues that the severe and prolonged irritation of the sensory nerves of the stomach exercises an inhibitory influence on the motor nerves of the intestine.

Cicatrices and adhesions that persist after the ulcer is healed may interfere with the functions of the stomach, and secondarily of the intestine, in a variety of different ways. The peristaltic movements of the stomach, for instance, may be hindered; the food may be retained for an abnormal time, and in this way gastritis, with all that it entails, be secondarily produced.

The state of the general health varies greatly. As I have mentioned above, there are many cases in which the ulcer remains latent for a long time, so that the patient complains of no symptoms whatever. In those patients who complain of violent pain, who suffer from frequent and abundant vomiting, who are afflicted with occasional attacks of hematemesis, the general nutrition must naturally suffer. It is due to these differences that we see some ulcer patients who look well and whose general nutrition is as good as could be desired, and others who are very anemic and very much emaciated. Medium degrees of anemia are very frequently seen in patients who have been afflicted with ulcer for a long time. However severe anemia and emaciation may be, the patients rarely present the cachectic appearance of carcinoma. I am not inclined to attach any fundamental importance to this difference, and I do not think that the differential diagnosis between ulcer and carcinoma can be founded on the appearance of the patients alone. It cannot be denied that ulcer patients, even though they may be greatly emaciated, though they may be very anemic as a result of repeated hematemesis, of frequent vomiting, and of repeated attacks of pain, generally look quite different from cases of carcinoma in the later stages.

Patients who have been afflicted with ulcer of the stomach producing severe symptoms naturally develop a variety of nervous disorders. They become irritable and depressed, frequently suffer from headache, vertigo, etc.

Occasionally anemia persists for a considerable time after the ulcer is healed. Dysmenorrhea or amenorrhea is also frequently observed, and is sufficiently explained by the many weakening factors that afflict the patient.

Fever is not a symptom of ulcer. If a patient with ulcer of the stomach develops a rise of temperature, we must assume that some complication exists; if the temperature rises suddenly and the patient collapses at the same time, we must think of hemorrhage. In these cases, however, the temperature rapidly drops again. The rise of temperature is due to the retardation of the flow of blood and a decrease in the radiation of heat. A sudden rise of temperature may also be caused by peritonitis or other complications.

An objective examination of the stomach reveals little that is positive; as a rule, the region of the stomach does not protrude nor show anything abnormal. Only in cases that are complicated by ectasy do we see protrusion of the gastric region. If there is stenosis of the pylorus, active peristaltic movements of the stomach may occasionally be seen.

On palpation, painful pressure-points will occasionally be found. This manipulation should be performed very gently. The absence of a tumor is frequently considered a valuable negative sign; this is undoubtedly true in the great majority of cases, particularly in relatively recent ulcers, for here we rarely feel increased resistance nor a tumor. In old ulcers, however, that extend to neighboring organs, whose base is in contact with these organs, and whose margins are thickened and swollen, this is not the case. Gerhardt in particular has furnished a very careful description of the tumor-like lesions that are seen in old ulcers. He distinguishes the following 4 varieties :

1. The ulcer itself may be felt; its base is thickened and plate-like, its margins hard. This form of palpable swelling is recognized by its flat, plate-like feel, its sensitiveness to pressure, and its permanence. These ulcers can be felt particularly well if they are situated in that small portion of the anterior wall of the stomach that is most readily palpable. In many instances an ulcer of this kind may even be felt through the left lobe of the liver. To judge from the statistics we have in regard to the seat of gastric ulcers, this peculiar resistance and swelling of the stomach-wall can be felt only in a very small proportion of ulcer cases. At the same time we must reverse the dictum that a palpable tumor always speaks against simple ulcer of the stomach, and must say, instead, that the presence of a small thin tumor speaks strongly in favor of ulcer in a patient who has suffered from stomach trouble uninterruptedly for three years or longer.

2. In a variety of affections of the stomach that are accompanied by spasmodic contractions of the organ and hyperacidity functional hypertrophy of the musculature in the region of the pylorus may lead to swelling of these parts, so that a tumor-like resistance can be felt. This is particularly frequent in ulcer of the stomach. The lesion may either be situated in the region of the pylorus and cause a swelling by its local effect, or it may be situated in some other portion of the organ and lead to the above-mentioned functional hypertrophy of the pyloric region by causing frequent attacks of cardialgia.

If the stomach is dilated and dislocated downward, this swelling can usually be felt very much better.

3. If perforative processes occur, a tumor-like mass of exudate or an encapsulated abscess may form and become palpable at the external aspects of the ulcer. In these cases we see a rapidly growing tumor develop after years of stomach trouble; at the same time the patients begin to fail, and the diagnosis of carcinoma will usually be made unless particular care is exercised.

4. Old large ulcers frequently involve extensive portions of neighboring organs (pancreas, left lobe of the liver, spleen). The portions of these organs that are involved form plugs that push their way into the ulcer; later they become chronically inflamed and hard. In this way massive painful tumors that may even grow slowly are formed. In the last two cases particularly the gastric contents should be analyzed. If an increase of hydrochloric acid is found, we are not apt to make so pessimistic a diagnosis (Gerhardt).

Reinhard has published a summary of 16 cases of ulcer of the stomach with tumors, and this report gives us a great deal of information in regard to the significance of palpable tumors in the diagnosis of ulcer or carcinoma. Among these 16 cases, in all of which the diagnosis of ulcer was corroborated by an autopsy, the tumor in 6 cases was found to be a hypertrophic enlargement of the pylorus due to cicatricial stenosis; in 6 other cases the result of adhesions between the stomach and neighboring organs. In a few of these cases the ulcer itself had extended to these neighboring organs. In 1 case of the 16 there was an abscess; in 3 cases foreign bodies (a mass of hair, chalk, vegetable material).

At all events, we may say that the formation of tumors is exceedingly rare in ulcer when we consider how frequent ulcer is.

The Analysis of Stomach-contents Removed through the Stomach-tube.—We frequently see the opinion expressed that the use of the sound is contraindicated in ulcer. We are told not to employ the sound in cases that have recently sustained a gastric hemorrhage, nor should we employ it in those cases in which the ulcer is situated in the cardiac region of the stomach. In general, however, there is no good reason why the sound should not be passed. I believe that the violent vomiting that is so frequently seen in ulcer, is more dangerous than the careful employment of a soft stomach-tube. The stomach-contents should not, of course, be aspirated, nor should the patient be allowed to express the stomach-contents. If the diagnosis "ulcer" is already positively established, the examination of the stomach-contents for further diagnostic corroboration may be omitted; but in all those cases in which the nature of the disease is doubtful aspiration of the stomach-contents should always be performed as in all other diseases of the stomach, for it is the only means at our disposal for determining the character and the intensity of the digestive perversions that may exist.

Formerly, it was universally believed that in all diseases of the stomach the functions of the organ were perverted in the sense of a reduction of their powers. In the case of ulcer the belief was generally prevalent that the functions of the stomach were performed with less energy than in normal subjects. In 1886, Ewald expressed himself as follows: In gastric ulcer the chemism of the stomach may be perverted in different ways. Simple circumscribed ulcer probably does not lead to changes in the secretion of gastric juice. Any abnormalities in the chemism of the stomach are essentially due to complicating catarrhal states of the gastric mucosa. This view has since been found to be incorrect, and Ewald himself has abandoned it. I was the first to demonstrate in a large number of ulcer patients that the secretion of gastric juice is greatly increased in ulcer; in other words, that there is usually hyperacidity. If the stomach-contents of an ulcer patient is analyzed after a test-breakfast or a test-meal, it will be found that the time of digestion is not prolonged, but, on the contrary, if anything, reduced. Quite frequently the stomach will be found completely empty three hours after a test-meal and one hour after a test-breakfast. In some instances the organ seems to get rid of its contents in even less time.

Chemical examination of the stomach-contents, as a rule, reveals high values for hydrochloric acid. It is impossible, of course, to draw a sharp line between normal acidity and hyperacidity. In one subject a certain value for the gastric acidity may be regarded as large; in another subject it may be considered normal. I found that the average acidity of 75 cases that I examined recently was 105 after a test-meal. The maximum of free hydrochloric acid that I have found in ulcer was 89. In these cases the total acidity was 130; the average for free hydrochloric acid in these cases was figured to be about 50, but we frequently found values over 60 even after a test-breakfast.

I wish particularly to insist that the results of the chemical examination of the vomit do not possess the same significance as the results of such an examination performed with stomach-contents that is removed at a definite time after a test-meal or a test-breakfast. Many of the errors that have been committed in this respect must be attributed to the fact that many authors examined the vomit without considering the character of the food that was eaten, nor the time that elapsed between the ingestion of the food and the vomiting, without knowing in many instances whether or not the patient had eaten anything in the meantime.

Any physician who has had occasion to examine many stomach cases knows that the analysis of the vomit always yields doubtful results, because so many sources of error exist. The only way in which to arrive at definite conclusions is to perform the examination of the stomach-contents in the manner outlined above.

We can readily understand, from what has been said in the section on the pathogenesis of ulcer, that there may be cases of ulcer, and that there are, in fact, such cases, in which the values for hydrochloric acid are not increased, for ulcer is not the cause of the increased hydrochloric acid production, but the latter condition predisposes to ulcer and impedes the healing of an ulcer that is already formed. This tendency to increased hydrochloric acid production may disappear, even though the ulcer persists. We frequently encounter old deep ulcers that never heal; as a matter of fact, there may be a condition of subacidity even in the presence of ulcer—for instance, in those cases where an ulcer develops on the basis of a carcinoma. Other catarrhal conditions or ectasy that leads to a prolonged retention of the food may also exercise an influence on gastric secretion. Nevertheless, we must regard it as the rule that ulcer is usually accompanied by hyperacidity. If hyperacidity is not present, however, we should not, therefore, conclude that the diagnosis "ulcer" is wrong, provided that all other symptoms speak for ulcer. At the same time we should always attempt to determine the cause of this abnormal condition.

I have already mentioned that ulcer and hypersecretion are quite frequently found together. I refer to the section on Hypersecretion for a more detailed description of this state. We need hardly mention that the presence of hypersecretion in ulcer, particularly if ectasy is also present, causes changes in the gastric contents, so that an examination of the stomach-contents under these conditions furnishes different results than in simple ulcer. The same applies to those cases in which stenosis of the pylorus with secondary ectasy develops. I refer to the different sections on these conditions for the details.

The quantity of urine is naturally reduced in ulcer whenever the ingestion of food is reduced and whenever there is much vomiting. No other characteristic changes are found in the urine. The acidity of the urine normally decreases in healthy subjects after each meal; the urine, in fact, may become neutral or even alkaline. Abundant vomiting of gastric juice that contains much hydrochloric acid naturally, therefore, reduces the acidity of the urine for several hours.

Gluczinski called attention to the fact that the chlorin in the urine is reduced particularly in those cases where there is a combination of great hyperacidity and impaired absorption. This fact cannot be utilized in the differential diagnosis between ectasy caused by carcinoma and ectasy complicated with ulcer. For a time it was believed that this was possible, but von Noorden and Stroh showed that in any stenosis of the pylorus, for instance, in which the absorption of food is reduced, the chlorin in the urine is also greatly reduced.

Complications and Sequelæ.—Ulcer may lead to a variety of complications and sequelæ. The most important of these are perforation, general and circumscribed peritonitis, adhesions, the formation of sacculated abscesses, stenoses, and the complication with carcinoma. Less frequent are severe pernicious anemia, distortion of the stomach, and general cutaneous emphysema.

Perforation.—The ulcer frequently extends downward to the serosa of the stomach; ultimately it involves neighboring organs and leads to the formation of adhesions between the stomach and these organs. It may happen that the serosa becomes perforated before solid adhesions with neighboring organs are formed. If this occurs, free perforation into the abdominal cavity results, and secondarily peritonitis develops. Just as pneumothorax develops whenever a cavity in the lung perforates into the pleural cavity before adhesions have formed between the two leaves of the pleura, perforation of an ulcer through the serosa before adhesions have formed allows the passage of air and stomach-contents into the free abdominal cavity.

Such perforations into the abdominal cavity occur almost exclusively in ulcers of the anterior wall of the stomach, and only in exceptional cases in ulcers of the posterior wall. This is explained by the fact that ulcers in the latter region, if they extend to the serosa, usually lead to adhesions with the organs in that portion of the abdomen.

Perforation may occur without any particular cause. In some cases, however, it seems to follow violent exertion. Occasionally it occurs during defecation or vomiting. The ingestion of a large meal, violence from without, a blow in the region of the stomach, etc. may all lead to perforation.

The symptoms are very characteristic. The patients suddenly experience a violent pain in the abdomen; some even state that they feel as if something had torn within them. The abdomen usually becomes rapidly distended and very sensitive to pressure, so that the slightest contact is painful. The liver and spleen dulness disappears as soon as air enters the abdominal cavity, and the diaphragm at the same time is forced upward.

The change in the condition of the patients is very rapid. They fall into collapse, they look very ill, the pulse is small and thread-like, cold perspiration breaks out, and the extremities feel very cool. Peritonitis itself causes few symptoms, and all the signs of this condition are usually masked by the general symptoms we have described. The temperature rises only a little, and may occasionally become subnormal. Traube was the first to call attention to the fact that vomiting is usually absent in cases of free perforation. The absence of vomiting may be considered an important diagnostic sign.

As a rule, perforation with the passage of gas and stomach-contents into the abdominal cavity leads to death in a short time—usually within a few hours. Occasionally, however, perforation seems to heal even though the symptoms are exceedingly violent in the beginning. Such a cure, however, is very rare, and is seen only if perforation occurs when the stomach is empty—that is, when no stomach-contents, but only air, enters the peritoneal cavity. I saw a case of this kind some time ago. The patient developed all the symptoms of perforation into the free abdominal cavity. The hepatic and splenic dulness disappeared, and there were a severe degree of meteorism, collapse, and the other symptoms of this condition. Nevertheless the patient recovered completely. In this case, too, the patient had taken no food for a long time before the perforation occurred. Hall has also described a case of this character that terminated favorably, and has gathered six other cases of a similar character from the literature. The reason why these cases are relatively so rare is that perforation rarely occurs when the stomach is empty, but usually when it is more or less distended by food and gas.

The symptoms of perforation, however, are not necessarily so violent. In some cases the abdomen is distended only very slightly or not at all.

If the ulcer slowly extends to the serosa, circumscribed peritonitis or perigastritis develops. Occasionally peritonitic friction-sounds may be heard if the ulcer is situated in the anterior wall of the stomach. In these cases adhesions frequently develop secondarily without causing any symptoms; in other cases again these adhesions may lead to a variety of disturbances. They seem particularly liable to interfere with the motor function of the stomach. The character of the disturbances will vary with the location of these inflammatory adhesions, and the involvement of this, that, or the other neighboring organ. It would lead us too far to consider all these possibilities; as a rule, perigastritis of this character can rarely be diagnosed with certainty. In many instances pain may be elicited on pressure in an area corresponding to the location of the inflammatory lesions. Rosenheim is correct in his statement that perigastritis may be the cause of pain in those cases where the presence of an ulcer is positively determined and where all treatment directed against the ulcer remains futile.

It may also happen that adhesions form with neighboring organs, and that later the gastric ulcer perforates directly into these organs. Brünniche describes a case in which the ulcer perforated into the heart; Habershon, another case in which the ulcer perforated into the transverse colon, and in which fecal vomiting occurred. West reports a case in which the ulcer perforated into the portal vein and in which, subsequently, fatal pylephlebitis developed. Perforation of gastric ulcers into the pericardium and the pleural cavity has been observed. Pick has described a case in which a round ulcer of the stomach perforated the abdomen. Here the perforation led to the formation of an abscess of the thoracic wall that finally eroded and destroyed several of the costal cartilages, so that the clinical diagnosis of a rapidly growing sarcoma of the ribs was made. Kolaczek[1] described a case in which gastric ulcer led to the formation of a diverticulum of the stomach. This lesion simulated a neoplasm that started from the wall of the stomach and became adherent to the abdominal walls. The patient was operated upon, and the diverticulum of the stomach was discovered. Before the ulcer perforated adhesions had formed with the abdominal wall, so that traction on the wall of the stomach was exercised by the adhesive bands.

It rarely happens that the ulcer perforates through the pleural cavity and leads to pneumothorax or pyopneumothorax.

Subphrenic Abscess; Pyopneumothorax Subphrenicus.—Ulcer rarely leads to the formation of an encapsulated abscess containing air underneath the diaphragm. Leyden was the first to describe such a lesion. He also originated the term that is in common use to-day, "pyopneumothorax subphrenicus." The first case that Leyden reported in 1880 was not diagnosed correctly during life. On autopsy it was found that an ulcer of the lesser curvature of the stomach had perforated the stomach-wall and had caused the lesion.

The clinical picture of these cases is essentially that of pyopneumothorax, but a number of typical symptoms indicate that a pus focus containing air is situated below the diaphragm.

Every case of pyopneumothorax subphrenicus does not necessarily originate from a gastric ulcer. Duodenal ulcers or other eroding processes of the intestine, perforation of the appendix, or abscesses of the liver may lead to the same condition. The majority of cases of left-sided pyopneumothorax subphrenicus, however, are caused by gastric ulcer. It is not necessary, of course,—and this undoubtedly makes the diagnosis more difficult,—that a large amount of air accumulate; in many instances we see the development of a circumscribed abscess underneath the diaphragm—that is, of a so-called subphrenic abscess. In these cases symptoms pointing to the accumulation of air are necessarily absent. The most important feature of the diagnosis is the demonstration of an exudate in the lower portion of the thorax; at the same time cough and expectoration are absent for a long time (Senator). It is important to determine that the lung above the exudate is intact and distensible, that its boundary extends downward on deep inspiration, and that there is pure vesicular breathing. The signs of increased pressure in the pleural cavity are absent at the same time, or are, at least, but slightly evident. The thorax on the afflicted side is, as a rule, not very ectatic; the intercostal spaces are scarcely obliterated. In those cases in which air and pus are present together, the lower portions of the thorax protrude very much. The heart is only slightly dislocated to the opposite side,—very much less so than in accumulation of pus and air in the pleural cavity. As a rule, the heart is pushed slightly upward. The liver, on the other hand, extends deep down into the abdomen; occasionally as far as the umbilicus or even still further. The lower margin of the organ can usually be distinctly palpated at this level. Manometry is also a valuable aid to the diagnosis of this condition. Pfuhl has called attention to the fact that the manometer rises on inspiration if the canula enters the cavity below the diaphragm, and that on expiration it falls. In case the lesion is within the pleural sac the reverse will be seen.

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[1] *Mittheilungen aus dem Grenzgebieten der Med. u. Chirurg.*, vol. i., No. 2.

There are, of course, many subphrenic abscesses that are so small and are situated in such hidden positions that they cause no objective symptoms. Occasionally, though rarely, the abscesses perforate through the diaphragm into the air-passages so that large quantities of pus are suddenly expectorated.

From all that we have said, it will be seen that subphrenic abscesses containing air are easily recognized if we remember all the different diagnostic criteria that we have described and that correspond essentially with those originally described by Leyden. The diagnosis of those subphrenic abscesses that do not contain air is more difficult, particularly if the inflammation extends through the diaphragm into the pleural cavity of the same side as the abscess, and if in this manner a pleuritic exudate of the same side develops. Senator[1] is undoubtedly right when he states that we are frequently justified in assuming that subphrenic abscess forms the connecting link between an ulcer of the stomach and pleurisy of the left side in those cases particularly where the former lesion precedes the latter. Senator mentions a number of other factors that aid in the diagnosis of subphrenic abscess complicated with pleuritis: (1) Violent pain, particularly in the epigastric region and in the hypochondriac region of one side; (2) pain and stiffness of the back when the patients attempt to sit up; (3) pain on belching and sobbing; (4) the fact that patients occupy the dorsal position even though there is a large pleuritic exudate; this is considered an important sign of subphrenic abscess, because, as a rule, in pleurisy patients lie on the diseased side; (5) more or less severe edema of the lower lateral and posterior portions of the thoracic wall extending into the lumbar region. None of these five symptoms alone demonstrates the presence of subphrenic abscess with any degree of certainty. It will be rare, however, to find all of them together in cases of pleuritic exudate or empyema, so that when we do find them, we are justified in suspecting the presence of subphrenic abscess.

Subphrenic abscess may terminate in different ways. We have already mentioned perforation through the lung with expectoration of abundant quantities of pus. The abscess rarely perforates into the abdominal cavity. When this occurs, general suppurative pleuritis or gangrenous peritonitis and rapid death ensue. Perforation through the skin is more frequently seen; here a gastrocutaneous fistula develops. A few cases are also on record in which pus perforated into the pericardial cavity, leading to secondary purulent pericarditis or pyopneumopericarditis. The pus has been known to perforate into the transverse colon. Here profuse diarrhea resulted, and an abundant quantity of pus was found in the stools. Occasionally inflammation of the portal vein and thrombosis have been known to develop. The pus may perforate into the pleural cavity, and if no adhesions were present, lead to pyopneumothorax or pyothorax.

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[1] *Charité-Annalen*, 1884.

We see, therefore, that under all circumstances the development of subphrenic abscess from ulcer is a serious complication. The only way in which the danger of this complication can be mitigated or possibly removed is by making an early diagnosis and giving the patient the benefit of surgical treatment.

A very rare complication of perforation of the stomach from ulcer is general subcutaneous emphysema. Demarquay[1] was the first to call attention to such cutaneous emphysema after solutions of continuity occurring in the stomach or the intestinal canal. He showed that perforation of the gastro-intestinal tract, followed by the entrance of gas into the free abdominal cavity, may lead to cutaneous emphysema if, at the same time, the parietal peritoneum is injured; as, for instance, after perforating traumata of the abdominal wall and the intestine, or traumatic lesions of the parietal peritoneum and the intestinal canal without perforation of the abdominal walls. Such an injury might be inflicted by some blunt instrument. His views are different from those of Roger,[2] for this author thinks that subcutaneous emphysema occurs only if the solution of continuity involves a portion of the intestinal tract that is normally in contact with the subcutaneous tissues—as those portions that are not covered by peritoneum—or is accidentally in contact with these parts (adhesions with the parietal peritoneum).

Poensgen[3] has given us a careful summary of all the cases of cutaneous emphysema following perforation of the intestine or the stomach into the free peritoneal cavity. He has also reported a case that he observed in Kussmaul's clinic. In the latter case, as in another instance reported by Korach,[4] the gas that escaped into the peritoneal cavity was combustible. In Korach's case there were a gastric ulcer and advanced ectasy. The ulcer perforated and gas escaped into the free abdominal cavity; two or three hours afterward circumscribed cutaneous emphysema appeared in the region of the umbilicus which soon extended with great rapidity over the whole body, until finally the patient died. The gas that escaped through a puncture in the skin, when lighted, burned with a puffy, non-luminous, slightly bluish flame; in other words, showed all the characteristics of a hydrogen flame. The perforation was preceded by a serious error in diet (eating an abundant quantity of bread and potatoes). This led to profuse fermentation and the formation of large quantities of gas, particularly of hydrogen, in the stomach. This in its turn led to rupture of the ulcer, digestion of the peritoneum, and finally general subcutaneous emphysema. Korach assumes that in this case the general emphysema primarily started from the subperitoneal cellular tissue of the anterior abdominal wall. He argues that the destruction of the parietal peritoneum was caused by the digestion of these parts by the stomach-contents that were poured into the peritoneal cavity; in this way gas entered the subperitoneal cellular tissue and passed from here through the intermuscular and interfascial cellular tissues, into the subcutaneous cellular tissue.

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[1] *Essai de pneumatologie médicale*, Paris, 1866.

[2] "De l'emphyséme généralisé," *Arch. gén. de méd.*, 1862.

[3] Poensgen, "Das subcutane Emphysem nach Continuitätstrennungen des Digestionstractus, insbesondere des Magens," *Inaug. Diss.*, Strassburg, 1879.

[4] Korach, *Deutsch.-med. Wochenschr.*, 1880.

The diagnosis of general cutaneous emphysema can readily be made from the great distention of the skin and the characteristic crepitation that is heard when the skin is palpated.

Other sequelæ of ulcer that must be mentioned are stenoses of the cardia and the pylorus. In general, ulcers are rarely located in the cardiac region of the stomach, and consequently cicatricial stenosis of this portion of the organ is rarely seen to follow ulcer. The ulcer is much more frequently located at the pylorus or in its immediate vicinity; consequently the termination of ulcer in cicatricial stenosis of the pylorus is much more frequently seen. In the same way perigastric adhesions in the neighborhood of the pylorus may lead to contraction or twisting of the stomach, and in this way to ectasy and relative insufficiency of the organ.

Other forms of adhesions may also interfere with the motor function of the stomach and lead to a variety of symptoms that result from this interference. Whether or not, as some authors assume, spasm of the pylorus caused by ulceration can lead to ectasy, is not decided. It is at least conceivable that frequently repeated spasms of the pylorus might lead to hypertrophy of the pyloric musculature and ultimately to a stenosis of the lumen of the pylorus. We can hardly doubt that muscular hypertrophies of this character are often produced by frequently recurring spasms. Leichtenstern has reported a case of tonic spasm of the cardia that ultimately led to hypertrophy of the esophagus. Hypertrophy of this kind, however, will only rarely lead to severe degrees of stenosis. We refer, for a detailed description of these secondary ectasies, to the sections on this condition.

Another sequel of ulcer that is not so frequent as those described above is severe anemia. Secondary anemia follows ulcer in the same way as it follows other chronic diseases of the stomach, as carcinoma, atrophic catarrh of the stomach, extended toxic gastritis, etc. This secondary anemia, according to Rosenheim, occasionally assumes the character of typical progressive anemia. We need not be surprised to find progressive anemia in diseases of the stomach in which the functions of the organ are permanently impaired, in which the general nutrition of the patient suffers to a great degree. Apparently, however, things are different in the case of ulcer, for here the function of the stomach *per se* is not impaired ; on the contrary, the powers of the organ are occasionally increased. If, therefore, we occasionally encounter serious anemia in ulcer, this must be explained from the insufficient amount of food that is eaten, from frequent vomiting or constantly recurring attacks of pain, and, above all, repeated hemorrhages. If in cases of this character the anamnesis does not indicate that an ulcer exists at the same time, if the hemorrhages remain undiscovered, it may easily happen that the anemia that appears is mistaken for a primary one.

Complication of Ulcer with Carcinoma.—It has frequently been found that carcinoma develops secondarily on the basis of an ulcer. Hauser has reported the most careful investigations on the histologic processes that occur when ulcer is converted into carcinomatous proliferation of tissues. His investigations revealed that the carcinomatous proliferation starts from the glandular tubules at the margin of the ulcer as soon as cicatrization occurs, causing enormous proliferation of the glands and a change in the character of their epithelial lining in the whole area of the cicatrix.

Rokitansky and Dittrich, in their day, called attention to the occurrence of carcinoma with ulcer, and suspected that the two conditions were etiologically related. According to Lebert, 9 of every 100 cases of carcinoma of the stomach are due to ulcer. Zenker goes so far as to state that the majority of cases of carcinoma of the stomach originate in ulcer. Rosenheim states that the proportion is as 6 is to 100. Clinically we are frequently enabled to demonstrate that an ulcer develops into a carcinoma if we will only observe the course of the disease with sufficient care. The symptoms, of course, will vary according to the seat of the lesion.

At first more or less pronounced symptoms of ulcer appear for some time—namely, cardialgia, acid vomiting, hematemesis, circumscribed painful areas, etc. These symptoms, as is so frequently seen in ulcer, may improve for a time, only to grow worse again. After some time, however, the disease-picture changes ; the patient loses strength ; his general appearance becomes bad ; he grows cachectic ; the pain no longer appears in so typical a manner nor so regularly as in simple ulcer ; the appetite is lost more and more ; emaciation and general loss of strength proceed rapidly. If we succeed at this time in demonstrating the presence of a tumor in the pyloric region, and if considerable ectasy of the stomach develops at the same time, we must think of carcinoma. Of course, we must never forget that functional hypertrophy of the gastric musculature or an indurated scar in the region of the pylorus may lead to the formation of a palpable swelling, and that this is not a rare occurrence in gastric ulcer. The duration of the stomach affection, even though it persists for many years, is of no value in the diagnosis. An ulcer that persists for years may lead to the formation of a tumor, just as well as a carcinoma that develops secondarily from an ulcer. The tumor, itself, therefore, indicates very little. The diagnosis may be directed in favor of carcinoma if the symptoms apparently remain stationary, but at the same time the general strength of the patient rapidly decreases and a tumor develops within a comparatively short time. Occasionally the size and shape of the tumor may give some information, for simple muscular hypertrophy of the stomach or a cicatrix of the organ usually leads to the formation of a uniform tumor that generally remains small; carcinoma, on the other hand, produces a tumor that frequently assumes an irregular shape and feels nodular. The latter also increases in size in the course of the disease, whereas the first-named form of tumor does not grow.

It might be thought that the question whether or not a carcinoma is present could easily be answered by an examination of the gastric secretion; we know that in carcinoma of the stomach free hydrochloric acid is, as a rule, absent, whereas in ulcer, on the contrary, there is usually an excessive production of hydrochloric acid. This criterion, however, is not valid. We know from clinical observation that in these cases of carcinoma the excessive production of hydrochloric acid persists for a long time; in some rapidly fatal cases hyperacidity has even been found until death. As a matter of fact, it seems to be the rule that in those exceptional cases in which free hydrochloric acid is found in carcinoma of the stomach the carcinoma will be found to have developed from a round ulcer. Sticker[1] was the first to call attention to this fact at the Sixth Congress for Internal Medicine.

Rosenheim attempts to explain this peculiarity by assuming that carcinomata that complicate ulcer grow for a long time near the bottom of the ulcer, so that the mucous membrane of the organ apparently remains intact for a considerable period of time. To judge from my personal experience, the duration of the affection plays a considerable rôle. I have examined a large number of cases in which the hyperacidity that accompanied the ulcer gradually decreased, so that finally there was subacidity and ultimately complete absence of free hydrochloric acid. In these cases I succeeded in diagnosing the development of cancer on the basis of an ulcer from the presence of a slowly growing tumor, the increasing cachexia, and the symptoms of ulcer that existed for some time before. The diagnosis was always corroborated by autopsy. In order to render such a diagnosis the cases must be kept under observation for a long time and the stomach-contents frequently examined. A single chemical examination of the stomach-contents has still less value here than such a single examination in other diseases of the stomach. If cases of this kind are studied up to their death, it will usually, though not always, be found that the secretion of hydrochloric acid gradually decreases. The determination of this gradual decrease is very important for the diagnosis, provided we know that an ulcer existed and we can determine that a tumor is developing rapidly.

[Professor Barling[2] expresses the belief that some French observers have unduly magnified the frequency with which cancer is grafted upon gastric ulcer. His observation and study lead him to disbelieve in the frequency of its occurrence. Hirschfeld, of Berlin,[1] discredits the view that gastric ulcer predisposes to carcinoma, and states the fact that out of 900 cases of cancer of the stomach reported in Vienna, only 5.6 per cent. appeared subsequent to gastric ulcer. He also calls attention to the wide distribution of gastric ulcer in certain districts wherein carcinoma is no more commonly seen than elsewhere. It is also to be noted that females, who are most prone to gastric ulcer, are less liable than males to cancer of the stomach. In cities where gastric ulcer is rare, as in Vienna, the number of women with cancer of the stomach is greater than in Hamburg, where gastric ulcer is more than twice as frequent.—Ed.]

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[1] See *Verhandl. d. VI. Cong. f. innere Med.*, p. 371.

[2] *Brit. Med. Jour.*, October 19, 1901.

Hour-glass Contraction of the Stomach.—We have already mentioned that ulcer of the stomach may gradually lead to cicatricial malformations, adhesions, and anomalies in the position of the stomach. So-called hour-glass contraction of the stomach, a condition that is occasionally congenital, may also result from ulcer. Nearly all the cases of hour-glass contraction of the stomach that have so far been observed were found by chance on autopsy; nevertheless I do not doubt that this anomaly could be diagnosed during life in the majority of cases if the patients were examined with sufficient care. The nature of this disease, as indicated by the term "hour-glass form," consists in a division of the stomach into two compartments by a cicatricial retraction of a portion of its wall. Of the two divisions, one appertains to the cardiac region of the stomach, the other to the pyloric region, and the two communicate with each other through an opening that may vary in size in different cases. Fig. 18, copied from the treatise by Schmid-Monnard,[2] represents a typical example of hour-glass contraction of the stomach.

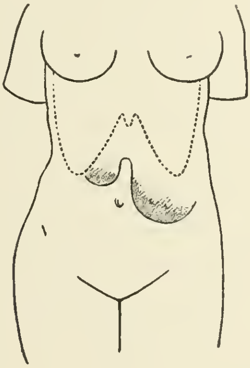

Fig. 18.

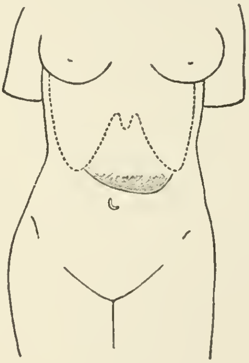

Fig. 19.

This case is particularly interesting because it constitutes one of the few instances in which the diagnosis was made during life, and in which the condition was cured by operative interference. Fig. 19

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[1] *German Congress of International Medicine*, 1902.

[2] *Münch. med. Wochenschr.*, 1893, No. 19.

represents the form of the stomach after the operation. The diagnosis of ulcer of the stomach, hour-glass contraction of the stomach, with incomplete stenosis, was made. The ulcer, together with the thickened, retracted portion of the stomach-wall, was excised, and the edges of the wound united so that the suture was situated vertically to the incision. By this operation the indentation of the stomach was completely removed, as was shown by inflation and percussion.

I need hardly mention that hour-glass contraction of the stomach may also be caused by other pathologic processes. Another important diagnostic feature may be mentioned in this place. Jaworski has reported a number of cases that he calls "ectasia ventriculi paradoxa." These, in all probability, were cases of hour-glass contraction of the stomach, for Jaworski mentions that he observed the following symptom, which he considers characteristic for this paradoxic ectasy—namely, that he was able to elicit succussion sounds, and still was unable to remove anything from the stomach through the sound; further, that he could fill the stomach with a stated quantity of water and was unable to remove more than a portion of the water he had introduced.

In one case reported by Eichhorst the deformity could be seen through the thin abdominal walls of the patient. Inflation of the stomach with carbonic acid gas or air is an important diagnostic adjuvant, for by this procedure the peculiar formation of the stomach can usually be recognized without difficulty, provided the abdominal walls are not too fat. In several cases I have been able to make the diagnosis of hour-glass contraction of the stomach by studying the outline of the organ after inflation. If this anomaly exists, both hypochondriac regions will protrude, while between the two hypochondriac swellings a furrow of varying depth will be seen. On both sides of this furrow there will be a loud tympanic sound, whereas this sound cannot be elicited in the middle. It may, of course, also happen that only one half of the stomach becomes distended. The size of the opening communicating these two halves varies greatly; Sacke[1] has reported a case in which the communicating orifice was only three-quarters of a centimeter in diameter. As a rule, the two halves are not equal; usually the pyloric half, as in the case pictured above, is smaller than the cardiac half. The food remains for some time in the latter, so that hypertrophy of the muscularis, and even secondary changes of the gastric mucosa, together with dilatation of this portion of the organ, may develop. We refer to the section on Anomalies in the Position and Form of the Stomach, for the other clinical symptoms of this condition.

The most common origin of this anomaly of form is ulcer. Usually the ulcer occupies both surfaces of the stomach; when it contracts it draws the middle portion inward; or there may be several ulcers on the anterior and posterior wall of the stomach that lead to contraction of the stomach-wall when they become cicatrized.

Incidentally, I might mention that this condition can be cured by operative measures, as shown by the case of Schmid-Monnard. It is, of course, impossible for me to describe the special surgical measures that are applicable to each individual case.

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[1] *Virchow's Arch.*, vol. cxxxiv.

Clinical Forms of Ulcer.—As we have already emphasized in describing the symptoms of ulcer, the clinical picture and the symptom-complex of this lesion may vary greatly. There are cases in which the typical picture of violent and regularly recurring cardialgia, with vomiting, occasionally hematemesis, circumscribed painful pressure-points, pronounced hyperaciditas hydrochlorica, and similar symptoms are seen. There are other cases in which the symptoms of severe anemia develop with mild, but in no wise characteristic, dyspeptic symptoms; finally, there are cases that remain altogether latent until the sudden occurrence of a hemorrhage or a perforation leads us to suspect the presence of ulcer. In view of the changeable character of the clinical picture many arbitrary types have been described, and, as a matter of fact, nearly every writer on this subject has enumerated a variety of clinical pictures of ulcer of the stomach and has grouped different forms together. At all events, in addition to those types that we have already described we may distinguish the following regular types:

1. Hemorrhagic Form.—This may run an acute or a more chronic course, and be latent in the beginning, so that the ulcer at first produces no marked symptoms. Suddenly, however, violent hematemesis occurs that may be so intense that the patients succumb to the profuse loss of blood. In other cases the hemorrhage is not immediately fatal, but as a result of the loss of blood severe anemia develops that, it appears, may exercise a favorable influence on the process of healing. We certainly occasionally see that the symptoms of ulcer rapidly improve and gradually disappear altogether after such a hemorrhage. Possibly hemorrhage *per se* is not responsible for this favorable issue so much as the fact that anemia develops; the patients are forced to remain quiet and to live on a carefully selected diet; in other words, a variety of factors are indirectly eliminated that interfere with the healing of the ulcer. In other instances the course of the disease is more chronic. Lebert has given this chronic hemorrhagic form a name that, in my opinion, is very well selected—namely, the "scorbutic form." The loss of blood during each attack of hematemesis is inconsiderable, but the hemorrhages recur very frequently. Bouveret reports the case of a patient who suffered from twelve hemorrhages in ten months. The stools almost constantly contained a small quantity of blood. The patient was so reduced and cachectic from this bleeding that a fatal issue was several times regarded as imminent; nevertheless the patient ultimately recovered, although the convalescence was slow.

[A case of this dangerous type of ulcer under my observation, that had continued for several years, was relieved by excision of the ulcer twelve months since, and has remained well to the present time. The lesion was located in the posterior wall, near the lesser curvature of the stomach, and the erosion had extended to the serous coat.—Ed.]

2. Acute Perforative Form.—Here, too, the ulcerative process runs a latent course. There are either no dyspeptic symptoms at all or only mild ones. Suddenly and unexpectedly perforation occurs that, in the majority of cases, rapidly leads to death. In almost all the cases the ulcers are situated on the anterior wall of the stomach. I have reported an example of this kind in the beginning of this section.

3. Chronic Dyspeptic Form.—Here the symptoms of dyspepsia are more conspicuous than the symptoms of ulcer proper. Judging from the former array of symptoms, the diagnosis of chronic catarrh may seem more probable than that of ulcer. Possibly the diagnosis nervous dyspepsia may be made. The patients, it is true, complain of pain and a feeling of discomfort after eating, but the pain does not recur regularly nor has it the character of cardialgic attacks. The region of the stomach may be very sensitive to pressure, and there may be an occasional attack of vomiting, but none of these symptoms are peculiar to typical ulcer, but are symptoms that are encountered in many other affections of the stomach. The most important points in these cases are the presence of hyperaciditas hydrochlorica and the absence of large quantities of mucus, for these two symptoms make it possible or probable that ulcer is present, even though they do not necessarily demonstrate its absence with certainty.

[The differential points above noted might incorrectly lead one to consider certain intractable cases of hyperchlorhydria as instances of gastric ulcer. The differentiation between these affections is occasionally trying, and must be settled by bringing to our assistance the discovery of localized tenderness and the other stigmata of ulcer.—Ed.]

4. Gastralgic or Neuralgic Form.—In this form gastralgic symptoms are the most conspicuous ones. It is possible to confound this condition with purely nervous cardialgia, and the differential diagnosis between the two diseases may frequently be very difficult. It is less difficult to differentiate the neuralgic form of ulcer from the attacks of pain of cholelithiasis.

5. Vomitive Form of Lebert.—In this form vomiting is the most disagreeable and the most serious symptom. The patients vomit nearly all food, and, as a result, emaciation is frequently very marked.

6. Cachectic Form.—The patients appear very cachectic; they are emaciated, very pale, and sick-looking, so that a diagnosis of carcinoma may be made. This form is seen chiefly in the late stages of an ulcer of the stomach of long standing. It is also seen in old cases of chronic hypersecretion that are associated with ulcer; also, finally, in cases of advanced ectasy that are due to ulcerative cicatrices. In this form the symptoms of general decay and of cachexia are the most conspicuous ones, although occasionally attacks of pain and vomiting may supervene.

These are the different types that Lebert first distinguished. Modern authors, as Rosenheim and Bouveret, still adhere to this classification. It would be an easy matter to distinguish other types, for in reality there are numerous intermediary forms. We must always remember, however, that the clinical picture of ulcer varies greatly; that the disease in one instance runs a typical course, in another produces no symptoms whatever, and that, finally, symptoms of the one or the other group may alternate. In order to avoid confusion the cases should be very carefully observed; but even if this is done it will occasionally happen that the diagnosis of round ulcer is made very late in the course of some disease that seems to be unimportant.

Duration and Course.—As indicated by the name "ulcus chronicum," ulcer of the stomach, as a rule, runs a chronic course. The ulcer may remain latent for long time, then a hemorrhage suddenly occurs, and the presence of an ulcer is revealed. At the same time it is usually altogether impossible to determine how long this ulcer has existed; in many cases it is also impossible to determine with certainty when the ulcer healed. As long as pain persists we must assume that the ulcer is not completely healed, or that, at least, sequelæ of the ulcer still exist. Even though the pain stops the ulcer may not be completely healed, but a recurrence may be in process of development.

There are, undoubtedly, cases that run a rapid course. The more recent an ulcer and the sooner it is rationally treated, the more readily will it heal. Many ulcers do not heal because conditions exist that are unfavorable to a cure; the patients, for instance, may continue to live on an inappropriate diet, and to eat coarse and irritating food. Such indiscretions will not only impede recovery, but will also favor the development and extension of the ulcerative process.

Lebert has determined that the average duration of an ulcer varies from three to five years. I do not believe that this statement is quite correct. A number of observations are on record of ulcer of the stomach that persisted for twenty or thirty years, and these observations are undoubtedly correct, for we know that many ulcers never heal completely, for the reason chiefly that they extend so deep into the stomach-wall and cover so large an area that a complete restitution to normal is impossible.

These cases of long duration that I have observed were usually complicated ones. If, for instance, an ulcer is situated in the pyloric region, the pylorus might become contracted by cicatricial tissue, and in this way ectasy of the stomach secondarily develop; or the ulcer might lead to adhesions with neighboring organs, with all the sequelæ that would result from such a complication.

The course of ulcer usually varies in many directions. If the patients are placed on a strict diet and if the general treatment is rational, all symptoms frequently disappear within a short time. The patients consider themselves cured; at the expiration of several weeks, months, or years, however, new disturbances suddenly appear that are analogous to the ones complained of before. Many of these cases may simply be a recurrence of ulcer, but whether this applies to all cases is doubtful. Sometimes the disturbances remain absent only so long as the patient adheres to the dietary regulations that have been given him. Regular cardialgic attacks, it is true, may disappear, but as soon as a slight variation in the diet is attempted, pain returns. Cases of this kind can hardly be considered cured. Occasionally all pain stops within a few days after the treatment of ulcer is inaugurated, particularly if the patients remain altogether quiet. If these patients were allowed to get up and to resume their former diet, the old disturbances would immediately return. We can speak of the cure of an ulcer with some degree of certainty only when all symptoms of ulcer have completely disappeared and when the patient has remained completely free from all distress for a long period of time. The only symptom that seems to persist, even after the ulcer has healed, is hyperacidity. This need not surprise us, for a tendency to hyperacidity is seen in many subjects; it is not a result of ulcer, but merely a factor that renders the ultimate cure of the lesion more difficult. In this sense hyperacidity may persist even after the ulcer is healed; even though this be the case, however, a new ulcer need not develop, for many persons suffer from hyperacidity who are not afflicted with ulcer. At all events, we should be very conservative in declaring a patient completely cured, and should advise all our patients to live a rational life in all respects for a long time after the complete disappearance of all symptoms of ulcer.

Prognosis.—In general it may be said that the more recent the ulcer, the more favorable the prognosis. The better insight that we have gained into the pathogenesis of ulcer has led to the adoption of more rational methods of treatment, so that nowadays the results are more favorable than formerly. We can readily understand, however, why, notwithstanding all this, many cases of ulcer are not cured; for many ulcers do not give rise to early characteristic symptoms, and, as a result, the patients are not treated correctly for a long time. Again, many patients, although they know that they are afflicted with ulcer, refuse to undergo a strict form of treatment so long as their symptoms are not very severe; many physicians even content themselves with prescribing a few general dietary regulations, or administering this, that, or the other drug. In this way many ulcers become chronic that would have been cured within a short time had a rest-cure been insisted upon from the very beginning. In hospitals and clinics the results of a strict rest-cure are much better to-day than they were formerly; in private practice a strict method of treatment for ulcer can rarely be carried out. This is in part due to external circumstances; nevertheless every physician should emphatically insist on a strict rest-cure. The sooner rational treatment of an ulcer is begun, the better the chances of a cure; the older the ulcer, the more difficult is it to cure; in addition, complications and sequelæ are more liable to occur. If the ulcers are very deep and extend to the serosa or neighboring organs, or if they occupy a large surface, they can never really be cured.

Relapses may, of course, occur, even though treatment is conscientiously carried out. We have already explained the reasons for this in the sections on the Etiology and Pathogenesis of Ulcer.

The mortality from ulcer is in general estimated as from 8 to 10 per cent. In each individual case the duration of the ulcer must be considered in rendering a prognosis. The more recent the lesion the smaller its extent and depth, and the sooner strict treatment is instituted the better the prognosis.

The seat of the ulcer is also important. Ulcers in the cardiac region are more liable to lead to stenosis of the cardia, and in this way to interfere with the ingestion of food. Ulcers in the region of the pylorus may lead to cicatricial stenosis of the pylorus with severe degrees of ectasy of the stomach. In these cases operative treatment alone offers chance of a cure. In cases of cardiac stenosis the methodic application of the esophageal bougie may do some good. If an ulcer undergoes cicatrization, this does not necessarily indicate that the patient is cured. Permanent disturbances may also result if the cicatrix is situated in other portions of the stomach than those mentioned above.

Deep ulcers may lead to fatal hemorrhage. Recently I had occasion to observe a case that died from a fatal hemorrhage from the pancreatic artery. Ulcers of the fundus may become adherent to the spleen, and in this way lead to splenitis and other serious sequelæ. If ulcers of the anterior wall are very deep and develop rapidly, perforation may occur. If ulcers of the posterior wall and the lesser curvature progress rapidly, lethal hemorrhages may result.

If hypersecretion exists together with ulcer, the prognosis as to general health is less favorable than if there is simple hyperacidity; for hypersecretion, while it may be somewhat reduced, is rarely completely cured. This perversion of function always leads to prolonged stagnation of the ingesta, and in this way constitutes a permanent source of irritation for the ulcer. I have already mentioned that carcinoma may occasionally develop from ulcer.

We see, therefore, that a patient with an ulcer of the stomach is exposed to manifold dangers. It is impossible in each individual case to render a favorable prognosis with any degree of certainty; we may say, however, that the prospect of cure will always be greater the sooner the patient undergoes a strict and rational ulcer cure.

Diagnosis.—The description of the symptoms that we have given shows very clearly how difficult the diagnosis of ulcer may be. In many instances the subjective symptoms alone enable us to make a diagnosis of ulcer with certainty; in other instances it may be impossible to recognize the disease, even though all the methods of examination that we know of are employed. We must never forget, on the one hand, that no single symptom, not even the vomiting of blood, absolutely demonstrates the presence of an ulcer; and that, on the other hand, an ulcer may, under certain circumstances, run its course without producing any symptoms whatever.

In attempting to render a diagnosis, three questions above all must be answered: First, Is an ulcer present? Second, if possible, Where is the ulcer located? Third, Is the ulcer a simple one or is it complicated, and what are the complications? If in the case of a young anemic girl violent attacks of cardialgia appear regularly one to two hours after each meal, finally terminating with vomiting of very acid masses; if the patient has a circumscribed painful pressure-point in the epigastric region, and possibly another one on the left side next to the spinal column; if the patient further gives a history of hematemesis a short time before—if, in a case of this kind, the analysis of the stomach-contents removed two or three hours after a test-meal or one hour after a test-breakfast shows little residue, but well-digested remnants and high values for hydrochloric acid, the diagnosis of "ulcer" may be made. A symptom-complex of this kind, however, is rarely observed. This, that, or the other symptom may be absent, or the majority of the symptoms may be absent and only one or the other be present.

Three symptoms are particularly important for the diagnosis: (1) Pain in the region of the stomach, that may either be felt in a circumscribed area on pressure or palpation, or may occur in paroxysms and be dependent on the ingestion of food. (2) Gastric hemorrhages. (3) Increased values for hydrochloric acid. All the other dyspeptic disturbances, as belching, heartburn, and vomiting, are less important. On the one hand, these last-named symptoms may be completely absent, and, on the other, they may be present in a great many other diseases of the stomach.

If the above-named three symptoms occur together, the diagnosis may be considered positive; if one or the other is absent, the diagnosis becomes doubtful. Quite frequently pain is the only symptom. If it is confined to one circumscribed area of the gastric region; if at the same time there is a circumscribed painful pressure-point next to the spinal column; if, finally the attacks of pain occur regularly at the height of digestion—the diagnosis of ulcer is the most probable one. It is also important to determine whether the pain varies in intensity according to the position of the body, and whether it is influenced by movements of the body. There are many cases in which the pain is only moderate, and in which no strictly circumscribed painful pressure-point can be found on palpation. This is particularly the case if the ulcer is situated in the posterior wall of the stomach. If in these cases an examination of the stomach-contents reveals hyperaciditas hydrochlorica, this finding speaks in favor of ulcer. At the same time increased values for hydrochloric acid do not by any means demonstrate the presence of an ulcer, for we must never forget that hyperaciditas hydrochlorica frequently occurs without ulcer. Patients with hyperaciditas hydrochlorica also frequently suffer from attacks of cardialgia, and in both diseases these attacks occur only after the ingestion of certain articles of food. Cardialgia in these instances is due to the increased production of hydrochloric acid, which, it appears, may lead to spasmodic contractions of the stomach. Other patients develop these attacks of cardialgia still more frequently; in fact, some of them seem to have them regularly as soon as they eat a large meal—particularly, therefore, after dinner. Some authors claim that the attacks of cardialgia that occur in simple hyperacidity differ in the time of their occurrence from the attacks of cardialgia seen in ulcer. This is not true; both are induced by the irritation of the ingesta; both occur at the height of digestion, and at a time when the maximum amount of hydrochloric acid is present in the stomach; and both terminate as soon as the ingesta have been completely expelled from the stomach.

It is true that cardialgic attacks in ulcer cases are, as a rule, more severe than those in cases of simple hyperacidity. It is also true that the cardialgia occasionally occurs somewhat sooner in ulcer than in hyperacidity. But these criteria are not decisive. I consider it much more important to remember that the cardialgic attacks occur more regularly in patients with ulcer than in cases of simple hyperacidity, for in the latter disease an attack may occur on one day and not reappear for several days thereafter.

Leube has advised the following therapeutic criterion in those cases where an ulcer is suspected but in which the diagnosis cannot be positively established (this applies to the cases we are discussing). He advises the patient to undergo treatment for ulcer, and then concludes from the result of this treatment whether or not the patient was afflicted with an ulcer. In making a differential diagnosis between ulcer and nervous cardialgia this method is certainly valuable and may be employed, and Leube recommends it particularly for such cases. In the cases we are discussing, however, it is useless, for in ulcer the hyperacidity is largely concerned in producing the cardialgic attacks. If patients with simple hyperacidity are put on treatment for ulcer, the attacks of cardialgia will stop. To judge from my personal experience, the chief difference between the two forms lies in the regularity with which the attacks of pain occur, for in simple hyperacidity without ulcer the attacks of pain do not occur so regularly as they do in ulcer; in hyperacidity, moreover, circumscribed painful pressure-points are absent. But even these distinctions are not always valid. In doubtful cases it is always rational to institute treatment for ulcer, for, as I have explained in discussing the pathogenesis, hyperacidity predisposes to ulcer, and is, in a sense, a preliminary stage of ulcer. The removal of hyperacidity is, therefore, an important prophylactic measure.

In other cases mild dyspeptic disturbances during the time of digestion appear; strictly circumscribed painful pressure-points cannot be found, nor do paroxysmal attacks of pain occur; then suddenly an attack of hematemesis occurs. Many physicians consider this vomiting of blood a positive sign of gastric ulcer, provided that carcinoma can be excluded. This symptom, however, may occur in other conditions, as I have explained in the symptomatology; carcinomatous and tuberculous forms of ulcer and simple hemorrhagic erosion may all lead to hemorrhage; in addition, hematemesis may occur in the initial stages of cirrhosis of the liver and in other forms of disturbance of the arterial and venous circulation, in varicosities, etc. Finally, it may happen that blood coming from some other organ, as from a ruptured varix of the esophagus, is swallowed and then evacuated by vomiting.

It is usually easy to avoid confusion between hematemesis and hemoptysis; as a rule, the blood in hemoptysis is foamy, light red, and mixed with air, and does not assume a dark color until later. It is evacuated by a coughing effort and the patient shows symptoms of pulmonary disease. Hematemesis is preceded by nausea, and the blood is dark. It may, of course, happen that a patient with hemoptysis swallows the blood and then evacuates it by vomiting, or, inversely, blood may enter the respiratory passages during an attack of hematemesis, cause irritation, and then be evacuated by coughing through the nose and the mouth at the same time. A careful examination of the patient, however, particularly after an attack; the fact that the sputum raised by subsequent coughing efforts contains blood and mucus; or, on the other hand, the passage of bloody stools after an attack, will frequently enable us to decide between the two conditions. A careful examination of the posterior nasopharynx will usually enable us to exclude hemorrhage from the buccal cavity.

It is much more difficult to differentiate between hematemesis caused by an ulcer and hematemesis that is due to some other cause—as, for instance, to hemorrhages that follow stasis in cirrhosis of the liver or in cardiac lesions. In disturbances of the portal circulation the veins of the esophagus in particular are dilated; these varicosities may tear or rupture, so that blood enters the stomach, is vomited, and in this way simulates a true gastric hemorrhage.

Hyperemia, even in the absence of ulcer, may also cause hematemesis. Watson has reported a case of this character that is frequently quoted. His patient was a woman who had suffered from gastric hemorrhage since her fourteenth year, the bleeding always occurring at the menstrual period. After she was married these attacks of hemorrhage did not occur during pregnancy and lactation, but appeared at all other times. Ewald also reported an interesting case of this kind.

In anemic subjects hemorrhages of this kind, even profuse gastric bleeding, are occasionally observed without ulcer symptoms. It is possible that in many of these cases a latent ulcer is present, but a number of cases of fatal hemorrhage have been observed in which nothing was found post mortem but a number of small hemorrhagic erosions of the gastric mucosa. [In rare fatal cases of hematemesis no lesion has been found post mortem.]

In hysteric patients gastric hemorrhages have frequently been observed both at the time of menstruation and at other times. In these instances prolonged observation alone can establish the diagnosis.

Small miliary aneurisms may also lead to such hemorrhages. The same applies to direct traumatic injuries of the gastric mucous lining. Heilbrunn[1] reports a case in which vomiting of blood occurred after drinking a glass of beer. The bleeding in this instance was caused by a small splinter of glass that was swallowed.

It is frequently difficult to recognize the primary source of these hemorrhages, and in general a careful examination of all the organs of the body will have to be made before a decision can be rendered. If in any given case hyperacidity can be demonstrated, the diagnosis will be more positive. Of course, a test-meal should never be given immediately after a hemorrhage in order to perform a quantitative determination of the acid in the stomach-contents. While I do not endorse the universally accepted view that the passage of the stomach sound is dangerous in ulcer, I certainly must warn against the use of this instrument immediately after a hemorrhage. The sound should not be passed for at least fourteen days after a hemorrhage.

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[1] *Centralbl. f. Chirurg.*, 1891, No. 6.

The diagnosis of ulcer of the stomach is difficult not only because the symptoms are latent in so many cases, but also because symptoms similar to ulcer appear in a large number of other diseases—for instance, carcinoma, ulcer of the duodenum, hemorrhagic erosions, gall-stone colic, and nervous cardialgia.

Carcinoma, in pronounced cases, is rarely confounded with ulcer; there are, however, cases in which it is difficult to decide whether we are dealing with an ulcer or a cancer. No single symptom should ever be considered conclusive for the one or the other condition; the only way in which to render a positive decision in difficult cases is to study the congeries of all the diagnostic points presented.

The absence of a swelling is in general considered to be an important negative sign of ulcer, and, inversely, the presence of a swelling usually suggests cancer. It is true that a tumor is, as a rule, absent in recent ulcers; in old ulcers, however, a swelling is quite frequently felt, particularly if the margins of the ulcer are hard and the base is thickened and plate-like. There may also be functional hypertrophy of the musculature in the pyloric region that may simulate tumor. The duration of the disease cannot be utilized in the differential diagnosis. It is true that carcinoma, as a rule, lasts for only one or two years, and that ulcer may persist for many years, but nevertheless the dictum of Gerhardt, "If an affliction of the stomach has persisted uninterruptedly for more than three years, a small thin tumor speaks greatly in favor of ulcer," can only be accepted with a certain reserve in individual cases. The small tumor may be due to an ulcer, but may also represent a beginning carcinoma. We know that carcinoma frequently develops on the basis of an ulcer. As a rule, it will be an easy matter to render a decision, at least in all those cases where we can observe the patients for some time; if, under these conditions, it is found that the ulcer does not grow, if the examination of the stomach-contents reveals that the peptic powers of the gastric juice are good, if the patient develops no cachexia, and if he seems to recover gradually under proper treatment, the presence of a small tumor will, of course, be interpreted in Gerhardt's sense; if, on the other hand, the tumor continues to grow, a cachectic condition develops and increases in severity, and an examination of the stomach reveals a progressive reduction in the secretion of hydrochloric acid, and at the same time the presence of lactic acid, the diagnosis carcinoma is the more probable one.

In rendering a differential diagnosis between ulcer and carcinoma ventriculi the examination of the gastric juice for hydrochloric acid, or better for free hydrochloric acid, and for its peptic power, is of fundamental importance. In the beginning many objections were formulated against the diagnostic significance of the determination of hydrochloric acidity; nowadays, however, it may be considered established that increased values for hydrochloric acid speak for ulcer, decreased values for carcinoma. I say, expressly, *speak for*, but not demonstrate, for hyperacidity may be present without ulcer, and subacidity or anacidity may be found in conditions other than carcinoma. If, however, the question is to be decided whether we are dealing with an ulcer or a carcinoma, the determination of hyperacidity on the one hand, or of subacidity or anacidity on the other, is important. It has been objected that, on the one hand, normal or even decreased values for hydrochloric acid have been observed in ulcer; and that, on the other hand, in exceptional cases no decrease in the hydrochloric acid of the stomach-contents has been observed in carcinoma; nevertheless these isolated findings do not impair the value of the observations reported above. Cases of this kind are exceptions, and are usually due to some particular influence that should be determined. The exception does not reverse the rule.

We may hold fast to the rule, that in doubtful cases pronounced hyperacidity indicates ulcer, decrease or absence of free hydrochloric acid indicates carcinoma. In some instances of incipient carcinoma that develop on the basis of an ulcer hyperacidity is encountered in the early stages, and is followed later by a gradual reduction in the hydrochloric acid of the stomach-contents. This, in my opinion, does not militate against the diagnostic significance of the HCl-acidity, but, on the contrary, speaks in its favor; for if ulcer is present primarily (that is, a disease that is accompanied by hyperacidity), and if secondarily a carcinoma develops (that is, a disease that is accompanied by a progressive decrease in the secretion of gastric juice), we must expect *a priori* that the prevalence of the one or the other disease will exercise an influence on the peptic powers of the stomach; in the beginning possibly we may find hyperacidity, but with the progressive development of carcinoma the condition of hyperacidity will gradually yield to one of subacidity, and finally to anacidity. Cases of this kind demonstrate how difficult the diagnosis may be in complicated cases, and at the same time demonstrate conclusively the value of the chemical methods of examination that we are discussing.

As an example of the value of these chemical examinations, I recently briefly reported the case of a man of fifty years, who consulted me in 1885.

The patient created the impression as though he was suffering from a carcinoma. He was extremely emaciated. He had been sent to the clinic with the diagnosis "carcinoma pylori, ectasia ventriculi." When the patient entered the hospital he was very cachectic, a tumor was felt in the pyloric region, and a considerable dilatation of the stomach determined. He suffered from frequent attacks of vomiting, from belching and other disturbances. We expected positively that the analysis of the stomach-contents would corroborate the diagnosis "carcinoma." It was found, however, that distinct reactions for hydrochloric acid (increased values for HCl) could be elicited, and that the peptic powers were good. The patient gained 10 kg. in a short time.

I saw the patient some years after this first examination; he had completely recovered and was free from all symptoms.

The course and the duration of the disease in this case demonstrated that it could not have been a carcinoma. The tumor was undoubtedly benign, and was due to an hypertrophy of the pylorus or to the cicatrix of an ulcer. If we had not examined the stomach-contents, we would certainly have made the diagnosis of carcinoma, like the physician who formerly had charge of the case.

I could enumerate many other cases from my own observation in which the correct diagnosis was only made by chemical analysis of the stomach-contents.

All other points in the differential diagnosis between ulcer and carcinoma are less important than the criteria we have mentioned. In any given case the facts that the patient is young, and that the general strength is well maintained, argue against carcinoma; occasionally, however, carcinoma is encountered in the early years of life, even in childhood, and in subjects whose general strength is unimpaired. The different criteria we have spoken of cannot, therefore, be utilized in certain individual cases.

The following case that I observed may be briefly reported as an instructive illustration of what I have said:

The patient was a widow of twenty-five years, who had been suffering from prolapse of the vagina since her last confinement; otherwise she was perfectly well. Six weeks before entering the hospital she began to suffer from anorexia, frequent belching, and occasional attacks of pain. On examination it was found that the subject was strong and well nourished, that there was an adipose layer of moderate thickness, and that the complexion was healthy; the abdomen was distended and the whole epigastric region sensitive to pressure. Circumscribed painful pressure-points, tumor, or succussion-sounds could not be determined. The boundaries of the stomach could not be clearly outlined. The first analysis of the stomach-contents showed that there was a weak hydrochloric acid reaction; later, free hydrochloric acid was found absent. Coarse and undigested particles of food in great number were always found in the stomach-contents. Meteorism gradually increased. Later, repeated attacks of vomiting supervened, during which coffee-ground masses were raised. At the same time there was obstinate constipation. Finally hydrops developed, but no real cachexia. The woman died six weeks after she entered the hospital. The clinical diagnosis was carcinoma ventriculi. The anatomic examination revealed a carcinoma of the pyloric portion of the stomach, that had originated from a chronic gastric ulcer.

In this case, therefore, nearly all the signs of carcinoma were absent. The vomiting of blood toward the end would signify either carcinoma or ulcer. The patient was young, strong, and showed no sign of cachexia. If the stomach-contents had not been analyzed in this case, the diagnosis carcinoma could not have been made. The fact that a slight hydrochloric acid reaction was elicited in the first examination may have been due to the development of a cancer on the basis of an ulcer. This case teaches us that the absence of cachexia, the youth of the patient, the good state of nutrition, do not justify us in excluding carcinoma. It also corroborates the observation that has frequently been made, that the course of cancer is particularly rapid in young individuals.

The following differential points between ulcer and carcinoma may be enumerated. In ulcer the tongue is red and moist as a rule, in cancer more frequently coated; in ulcer the appetite is good provided the patients do not refuse food for fear of the pain, in carcinoma the appetite is greatly reduced. In the latter case we frequently encounter a pronounced aversion to meat. Pain proper is frequently absent during the whole course of a carcinoma, or there may be only a dull feeling of pressure; in ulcer pain is the rule, and it usually occurs synchronously with the period of digestion; if vomiting occurs in ulcer, it usually appears early,—that is, one or two hours after eating; in carcinoma, on the other hand, it usually appears later, at less regular and at longer intervals.

The appearance of the vomit usually varies. In ulcer there is rapid digestion of the food, so that the vomit is well digested and there is little of it; as a rule, it is a fine pultaceous mass in which coarse remnants of meat are only exceptionally found. The vomit also usually gives a distinct Congo reaction. Only in those cases in which ulcer is complicated by hypersecretion are large quantities of food, consisting largely of amylaceous material, vomited. In carcinoma the quantity of vomit is, as a rule, large; many coarse and undigested meat fibers and other coarse particles of food are contained in it. The vomit has an acid odor, and chemical examination usually reveals the absence of free hydrochloric acid, but the presence of abundant quantities of organic acids, chiefly lactic acid. The vomiting of blood is less decisive in the differential diagnosis in carcinoma; it is true the amount of blood vomited is rarely so large as in ulcer; in the former condition, however, blood may be vomited for a much longer time.

These are the most important points that must be considered in rendering a differential diagnosis between carcinoma and ulcer.

A second disease with which round ulcer of the stomach may possibly be confounded is ulcer of the duodenum. In the majority of cases it is impossible to recognize an ulcer of the duodenum with certainty, or rather to differentiate it from an ulcer of the stomach. It is to be expected *a priori* that an ulcer situated in the neighborhood of the pylorus, in the first portions of the duodenum, must produce similar symptoms to ulcer of the pylorus itself. The following symptoms are of value in the differential diagnosis: In ulcer of the duodenum the pain is felt in the region of the right parasternal line; in ulcer of the pylorus it is found more toward the median line. If hemorrhage occurs in duodenal ulcer, the blood is usually evacuated through the bowels. The pain in duodenal ulcer, as a rule, appears later than the pain in an ulcer of the stomach; it appears to me that attacks of pain are frequently altogether absent in ulcer of the duodenum; dorsal painful pressure-points are also absent. It has not been determined whether or not hyperacidity also occurs in ulcer of the duodenum. I have succeeded in finding only one direct statement in this respect in Leube's work. This author reports a case of ulcer of the duodenum that terminated fatally from hemorrhage and in which the value for hydrochloric acid equalled 0.16 per cent., in which, in other words, there was no hyperacidity. Bouveret makes the statement that hyperacidity cannot be utilized in rendering a differential diagnosis, because it is one of the pathogenetic conditions both of ulcer of the duodenum and of ulcer of the stomach. He does not state whether this assumption is based on any direct investigations.

Ulcer of the duodenum occurs with comparative frequency after burns of the skin, and is more frequently seen in men than in women. In many of the cases there is a history of alcoholic abuse. All these factors, however interesting they may be *per se*, are of no value in rendering a diagnosis in an individual case. One factor that may possibly be of value in doubtful cases is the absence of vomiting in ulcer of the duodenum. The complication of duodenal ulcer with icterus is so rarely seen that it has no significance in the differential diagnosis. Icterus appears in those rare cases in which the ulcer extends to the orifice of the common duct, and where the latter is involved in the process.

We see from all this that it is very difficult to make a differential diagnosis between ulcer of the duodenum and ulcer of the pylorus. The most important points in this diagnosis seem to be the absence of vomiting and of hematemesis, the evacuation of blood exclusively downward, and the occurrence of attacks of pain, the latter appearing much later after a meal than the pain in gastric ulcer. In addition we know that ulcer of the duodenum pursues a latent course for a much longer time than ulcer of the stomach. Frequently the first symptom of an ulcerative process in the gastro-intestinal tract is the sudden appearance of a profuse quantity of bloody material in the stools and the sudden occurrence of severe anemia. In any given case, of course, it will be impossible to exclude ulcer of the stomach from these symptoms, but the possibility of duodenal ulcer should at least be suspected.

Ulcer of the stomach may also be confounded with hemorrhagic erosion of the gastric mucosa. A great number of cases of lethal gastric hemorrhage have been reported in which no ulcer was found, but merely hemorrhagic erosions. It is another question whether the latter lesions can be diagnosed at all, whether they can produce a distinct clinical symptom-complex. Until quite recently hemorrhagic erosions have been considered a rather frequent pathologic-anatomic finding, but nothing more. Einhorn,[1] and subsequently Pariser,[2] attempted to delineate a clinical disease-picture to correspond to this finding. Although it has never been shown that the picture described by these authors is really caused by hemorrhagic erosions, I think that it merits passing mention. No autopsies have so far been made in these cases, so that a positive proof cannot be forthcoming. The most important symptoms of hemorrhagic erosion, according to these authors, are burning pains in the whole gastric region ; the patients eat less than normal and become emaciated. As in ulcer, the chief complaint in hemorrhagic erosions is the pain, the attacks of pain following the ingestion of food, whatever the character of the diet may be ; they usually appear from a quarter to three-quarters of an hour after the meal and last about two hours. They are not, however, limited to any one definite location of the stomach, as in ulcer, but extend uniformly through the whole organ. In addition, dorsal painful pressure-points are never found. Pariser attaches particular importance to the character of the pain. According to him, it is burning, hot, and consuming, not gnawing, boring, or biting, as in ulcer. It appears to me that it is still more important to remember that pressure or a change in the position of the body does not influence the pain in any way,—that is, neither increases it nor relieves it. This is particularly striking when we remember that hemorrhagic erosions *per se* can produce violent pain, but that the latter remains uninfluenced by the manipulations that I have mentioned above.

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[1] *Berlin. klin. Wochenschr.*, 1895, Nos. 20, 21.

[2] *Med. Revue f. innere Med. u. Therap.*, 1897, No. 1.

Whereas Einhorn in his cases reports that the pain was moderate in degree, Pariser claims that the pain in his cases was almost insupportable. Occasionally there is a tendency to vomiting, although it rarely occurs. Einhorn found the chemism of the stomach different in different cases; in some instances it was reduced, in others normal. An increase in the hydrochloric acidity was found in only one case. Pariser found normal acidity in two cases, moderately reduced acidity in one. Both authors attach the greatest importance to an examination of the stomach-contents after fasting. They always found several small pieces of gastric mucosa in the wash-water. The microscopic examination of these shreds of mucosa revealed no pathologic changes excepting a proliferation of the cells in the interglandular tissues. Pariser also reports that the wash-water was always tinged slightly red from the admixture of blood; Einhorn, on the other hand, observed this in only one of his cases.

While we must recognize that the disease-picture we have described above presents certain peculiarities, we do not feel justified in considering it a distinct clinical entity: first, because so few cases have been recorded that presented this syndrome; second, because the clinical picture has never been verified by autopsy. Particles of mucosa are occasionally found in the wash-water in other diseases of the stomach. The character of the pain, it appears to me, being a purely subjective symptom, is of small importance. The absence of hyperacidity in the majority of cases of hemorrhagic erosion seems to me to be more important for the differential diagnosis; at all events it will be necessary to study many more cases of this character before we can undertake to diagnose hemorrhagic erosions of the stomach with any degree of certainty.

Another disease with which ulcer might be confounded is cholelithiasis. In the majority of cases the diagnosis is easy. The following points above all are important: The pain from ulcer appears regularly and at a definite time after eating. The pain in gall-stone colic also appears in paroxysms, but independently of the meals and without regularity. The chief painful pressure-point in cholelithiasis is found in the region of the gall-bladder. If the distended gall-bladder can be felt, and if a mild degree of icterus appears, the question may be considered settled; but icterus even of mild degree is frequently absent. If the liver is found enlarged and painful, or if the margin of the organ can be felt and is painful, this indicates gall-stone colic. [A peculiar tongue-like enlargement of that portion of the right lobe of the liver lying over the gall-bladder, the so-called "process of Riedel," is, when present, strongly indicative of cholecystitis.—Ed.] Attacks of cholelithiasis, as a rule, last longer than ulcer pain; the intervals between the attacks are usually longer in the former condition. The attacks of cholelithiasis usually appear suddenly, while the patient is enjoying the best of health; they are usually very severe and rapidly produce symptoms of collapse. The pain of ulcer hardly ever develops suddenly. The pain of gall-stone colic usually radiates toward the right, particularly toward the shoulder; slight febrile movements frequently accompany the attacks. If all the points that we have enumerated are insufficient to render a diagnosis, the stomach-contents should be aspirated and examined. Occasionally the vomit can be used for diagnostic purposes, but much will depend on the time when vomiting occurs. If examination of the stomach-contents or of the vomit reveals hyperacidity, this may be considered an argument in favor of ulcer of the stomach, although it is hardly a positive criterion.[1]

Ulcer of the stomach may also be confounded with purely nervous types of gastralgia. An important point in the differential diagnosis is the appearance, as a rule, of general nervous symptoms in addition to local ones. The symptoms of nervous gastralgia, moreover, differ in many respects from those of ulcer. The appetite, for instance, in nervous gastralgia is usually variable and irregular; occasionally there is a craving for particular articles of food. The attacks of pain by no means occur regularly after eating or at definite times. Occasionally the pain is relieved by pressure exercised over the stomach. Hematemesis is never seen in nervous gastralgia. Hyperacidity may be found in nervous gastralgia, but the values for the acidity of the stomach-contents are usually variable and are quite frequently normal.

While it may be difficult to make a differential diagnosis between purely nervous gastralgia and ulcer at once, it is, as a rule, an easy matter to do this if the patient is observed for a sufficiently long time. Leube recommends the employment of the electric current during digestion, and claims that disappearance of the pain on application of the anode in particular speaks for gastralgia. If the pain persists, either gastralgia or ulcer may be present. A positive finding alone,—that is, the disappearance of the pain during the time that the electric current is being applied,—can be considered of value in rendering a diagnosis.

Ulcer of the stomach should not be confounded with intercostal neuralgia. A careful examination will always enable us to differentiate between the two without difficulty.

Penzoldt calls attention to the fact that a movable kidney may occasionally produce the picture of an ulcer of the stomach, even including the hyperacidity. The only symptom that is absent is hematemesis. He claims that in practice the two conditions are frequently confounded.

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[1] Sticker has found, and I can corroborate his finding, that hyperacidity occasionally occurs during attacks of cholelithiasis.

To judge from my personal experience, it is, as a rule, an easy matter to avoid such an error if the patient is carefully examined, particularly by bimanual palpation. That hyperacidity frequently occurs together with movable kidney need not surprise us, for both forms of disease are relatively frequent. Movable kidney *per se* may produce certain symptoms for a time, but these, in my experience, have very little resemblance to the symptoms of ulcer.

If the diagnosis ulcer has been positively made, the second task is to determine the seat of the lesion. This can be done in only a small proportion of the cases. The clues that we possess for determining the seat of an ulcer are rarely absolutely reliable. Gerhardt has expressed the opinion that the diagnosis of ulcer is never positive unless the seat of the lesion can also be determined. Of course, the diagnosis in the latter case would be more positive; but if we made the diagnosis ulcer only in those cases in which we could determine the seat of the lesion with certainty, many cases of ulcer would undoubtedly remain unrecognized. I need only refer to the symptomatology of ulcer in order to refute Gerhardt's postulate. At the same time we are frequently enabled to determine the seat of the ulcer, and we are indebted particularly to Gerhardt for calling attention to many of the points that aid us in doing this. According to this investigator, pain on pressure and a tumor indicate that the ulcer is situated in the anterior wall of the stomach; pain in the back and hemorrhage, that it is located in the posterior wall. The location of the pain, and exacerbation of the pain whenever the patient occupies a lateral position, frequently enable us to make a differential diagnosis between ulcer of the fundus and of the pylorus. If the stomach is dilated, this always indicates ulcer of the pylorus or the duodenum. If the ulcer is situated in the fundus and becomes adherent to the spleen, splenitis may develop, and this may lead to chills that Gerhardt observed in three cases. Hour-glass contraction may cause double respiratory râles of a coarse caliber (Gerhardt). All these points are without doubt important, but in many cases they are not observed. I agree with Leube when he says that the seat of the ulcer can be diagnosed only in exceptional cases. Leube, it appears to me, is correct when he attaches particular importance to the following symptoms: If pain underneath the xiphoid process occurs regularly on swallowing solid cold or hot food, during the last act of deglutition, the diagnosis of ulcer of the cardia seems almost positive; at all events the appearance of pain in this location should warn us not to pass the sound for diagnostic purposes, as hemorrhage may readily occur. In this form painful spasms of the esophagus and regurgitant movements are occasionally observed. If cicatrization of the ulcer occurs, symptoms of stenosis of the esophagus are very liable to appear.

The third point that must be determined in rendering the diagnosis is the presence or absence of complications. The latter are less frequently seen in recent than in old ulcers. It is a well-known fact that complications are frequently found in the latter. All the symptoms of a recent ulcer may, however, be presented, and complications still be present. The most frequent and the most important of these complications is dilatation of the stomach. Formerly, when the chemism of the stomach in ulcer was unknown, it was usually assumed that in ulcer there was more or less catarrh of the stomach. Careful analysis of the stomach-contents has shown this view to be erroneous. Ectasy is more frequently encountered than catarrh of the stomach; as a rule, this condition develops after ulcer has persisted for some time. Many authors are inclined to assume that the appearance of symptoms of dilatation of the stomach in the course of an ulcer necessarily indicates that the ulcer is situated in the pyloric region or in the duodenum. In this general form this assumption is not correct. The symptoms of ulcer frequently occur together with symptoms of hypersecretion, as well as with those of hyperacidity. In cases of this kind ectasy is frequently present before ulcer appears. This ectasy may be the result of a cicatricial stenosis; if this is the case, however, the symptoms of stenosis of the pylorus must be presented. Or ectasy may be the direct result of hypersecretion even though no stenosis exists. I refer to the section on hypersecretion for the origin of this form. In all those cases, therefore, in which we determine that ectasy and ulcer exist together we should always think both of cicatricial stenosis of the pylorus and of hypersecretion, for either one of the latter conditions may be the primary cause of ulcer and of ectasy. All other possible complications are of small interest. We have already referred to them in discussing the course and the sequelæ of ulcer. They are chiefly adhesions, distortion of the stomach, etc. I merely refer in this place to what has been said above.

Treatment.—The process of healing in ulcer of the stomach is the following: At first inflammatory proliferation of the connective tissue at the base of the ulcer occurs, followed by gradual contraction that finally leads to cicatrization. The latter can only occur if the ulcer remains small and involves a limited area of the gastric mucosa; if it extends deep down into the tissues and covers a large area, healing by cicatrization is impossible. All treatment should be directed toward aiding this process of healing.

A deep wound in any portion of the external surface of the body will naturally be treated by sparing the affected region, by putting the wounded parts at rest. A wound cannot heal, or at best heals with difficulty, if it is continuously irritated by movements, by chemic, thermic, or other agents; for this reason the diseased portion of the body is placed at rest and all external injuries are avoided. The laity does not consider that a wound of the stomach should be treated in the same way; in other words, that rest and protection are the fundamental conditions of healing. Even physicians have failed to recognize that the rest-cure of ulcer should occupy the first place in the treatment of this disease. Even nowadays many physicians attempt to devise methods of treatment that permit the patients to follow their usual occupations, and to pursue their ordinary mode of life with only a few limitations, while they are undergoing a cure. Any attempt to cure an ulcer by this means must be considered very uncertain.

The laity in general is surprised to find that ulcers of the stomach resist treatment for so long a time; but, may I ask, What form of external ulcer would heal if it were continuously stretched and irritated, if it were continuously exposed to chemical, mechanical, or other irritants? It is true, of course, that certain conditions obtain in the stomach that do not obtain in the case of external wounds. It is also true that the acid of the stomach exercises a certain influence on the healing of the wound, or rather can be made responsible for the fact that ulcers do not heal readily. This should teach us to attempt to create ideal conditions for healing in the case of ulcer of the stomach, and to apply the same rules for curing an ulcer that we follow when we treat other wounds. The fundamental conditions, as I have said, are the greatest possible rest and the avoidance of all irritation.

The necessity of instituting a "rest-cure" has been recognized for some time. Cruveilhier postulated complete rest for the stomach as the fundamental condition of a cure of ulcus ventriculi. In England, Wilson Fox, B. Forster and Williams in particular, recommended the rest-cure. In Germany, Ziemssen and Leube have been particularly active in advising this method of treatment. The reason why this mode of treatment has failed to earn universal recognition is that it is more difficult to carry out a rest-cure in the case of the stomach than in any other organ or parts of the body. It is easy to protect the external portions of the body from irritation and to keep them at rest; in the case of the stomach this is very much more difficult, for the reason chiefly that it is almost impossible to place it at rest functionally for a prolonged period of time.

If the *indicatio causalis* of the treatment of disease is to remove the causes that produce the disease, this indication can hardly be fulfilled in ulcer of the stomach, for even if we assume that reduced alkalinity of the blood following circulatory disturbances on the one hand, and abnormally increased acidity of the gastric juice on the other, play an important rôle in the genesis of ulcer, even though we consider it empirically established that ulcer is particularly frequent in anemia and chlorosis, all these factors can hardly be considered the primary cause, but only predisposing elements. Ulcer is seen in only a small proportion of the many cases of chlorosis and anemia that are observed; we must assume, therefore, that some other factor is added to anemia and chlorosis in order that an ulcer may be produced. In the same sense we frequently see patients in whom the acidity of the gastric juice is increased, but who nevertheless do not develop ulcer. Again, an ulcer may occur in a perfectly healthy subject following trauma, a blow, etc. In order that an ulcer be formed, even in the cases that are predisposed to this lesion, and in whom the soil, so to say, is prepared for an ulcer, some direct injury must be sustained. The primary cause of the ulcer in the majority of cases acts only once, and usually long before the patient comes to us for treatment. For these reasons it is almost impossible to remove the causal indication,—that is, the primary factor that is directly responsible for the development of an ulcer. Treatment, however, can be instituted indirectly against this causal indication; in other words, we can proceed prophylactically. As I have explained above, it is not surprising to find that ulcer develops after a trauma of the stomach or after thrombosis of certain blood-vessel areas. This is due to the fact that the involved areas are no longer properly nourished, and consequently die. We see similar conditions in other portions of the body. The only surprising feature in the case of ulcer of the stomach is that the wound of the stomach heals with such difficulty and has a tendency to extend; we must endeavor, therefore, above all, to determine why gastric ulcer heals with such difficulty. We are inclined to see the chief cause, though not the only one, for this peculiarity in hyperchlorhydria.

If this assumption is correct, we should endeavor to combat this hyperchlorhydria with all the means at our disposal. If we succeed in remedying this perversion of function, then we create much more favorable conditions for the healing of an ulcer after any injury that the stomach may sustain. For this reason we must attribute a certain prophylactic significance to all treatment that is directed toward the correction of hyperchlorhydria.

We can proceed prophylactically in still another direction, although here, again, the effect is indirect. It is established that round ulcer occurs with particular frequency in chlorotic and anemic subjects. Daettwyler has demonstrated that general anemia is an abnormal condition that renders the healing of artificially produced ulcer difficult. This investigator determined that of two ulcers of equal size that were artificially produced in two dogs, the one healed in eighteen days in a normal animal, whereas the other one was not healed at the expiration of thirty-one days, if the dog were rendered anemic. This demonstrates that severe anemia renders the cure of an ulcer difficult, and it also demonstrates that we must combat anemia, for we know that ulcer heals with greater difficulty in anemic subjects than in persons who are not anemic.

I have mentioned in discussing the etiology of ulcer that the frequency with which ulcer occurs in different regions also varies greatly. Some authors have attributed the rare occurrence of ulcer in certain regions to the fact that certain parts of the population live on a vegetarian diet exclusively, so that more potassium enters their blood than in people who live on a mixed or an abundant animal diet. If it could be demonstrated that this is really the case, and that the introduction of potassium salts impedes the formation of ulcer, this point could be utilized with profit in the prophylaxis of ulcer; unfortunately this hypothesis has not been verified.

Rasmussen has stated that lacing produces circumscribed atrophy of certain portions of the gastric mucosa, just as it produces atrophic lacing-furrows in the liver. He argues that this produces necrosis from pressure and ulcer of the stomach. If this statement were correct, one of the most important prophylactic rules would be to combat energetically the universal habit of lacing.

From all that we have said we learn that there is really no prophylaxis proper of ulcer. The different injurious agencies that predispose more or less to ulcer can usually not be removed; some of them can possibly be remedied, and, as a matter of fact, we proceed against them as best we can.

The *indicatio morbi* postulates that conditions should be created in the stomach that will favor healing of the ulcer. The main condition is to avoid all irritation of the organ. The only way in which to do this is to stop the administration of all food by the mouth for some time. In this way even the irritation that is produced by the secretion of hydrochloric acid is removed.

While it appeared self-evident that placing the stomach at rest would be beneficial in ulcer, attempts to carry out this treatment have only been performed within the last few years. As was to be expected, the results of this procedure were very favorable. Up to a short time ago the administration of all food by mouth was interdicted only for a short time immediately after a hemorrhage. Donkin[1] was the first to treat a large number of cases of ulcer by rectal alimentation. He extended this treatment over twenty-three days and observed favorable results throughout. Call Anderson[2] and Boas[3] also reported very favorable results from exclusive rectal feeding in cases of severe and frequently recurring ulcers in which all other methods of treatment failed.

If it is at all possible, I am in the habit of beginning the treatment of ulcer by instituting rectal feeding exclusively for several days.

I do this not only in old chronic cases, but also in recent ulcers. In the latter class of cases, particularly, I have seen very striking results, the vomiting, the attacks of cardialgia stop within a short time, and the ultimate healing of the ulcer may be looked forward to with a greater degree of certainty in recent ulcers than in chronic ulcers if an absolute rest-cure is instituted. I believe that the treatment of all cases that present pronounced ulcer symptoms when they are admitted to our clinics and hospitals should be begun in this way.

The patients receive from two to four nutritive enemata *pro die.* At the expiration of six to eight days, or still later, a little food may be introduced by mouth. I need hardly insist that the patients should remain in bed. In order to quench the thirst I advise giving small pieces of ice, that the patients allow to melt in the mouth; in addition the mouth may be washed out frequently. Carlsbad water or salts that are usually employed with benefit in the subsequent treatment of ulcer, should, of course, not be administered during the period of exclusive rectal alimentation. In cases in which violent gastric hemorrhages occur, and in those cases that develop violent paroxysms of cardialgia and that suffer from very obstinate vomiting, exclusive rectal alimentation is, of course, to be preferred to all other methods of treatment. I need hardly modify this in detail.

Whether the treatment of a case of ulcer is begun in the way

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[1] *The Lancet*, 1890.

[2] *Brit. Med. Jour.*, 1890.

[3] *Diagnostik u. Therap. d. Magenkrankheiten.*

I advise or not, the diet remains the most important factor in the subsequent treatment of the case. Other factors in the treatment must not, of course, be neglected. Generally speaking, treatment nowadays is carried out according to the same principles that Leube and Ziemssen established in their so-called rest or ulcer cure. This treatment consists —(*a*) in the administration of a very digestible diet that should be liquid at first and uniform, and later may be more solid and mixed; (*b*) in the administration of certain mineral waters; (*c*) in keeping the stomach quiet, particularly during the first period of the treatment. The application of moist warm or hot compresses aids the cure.

The diet should be selected so that the food that is administered is as little irritating as possible. The best plan, if it can be carried out, is to refrain from the administration of all food by mouth for some days; after that a very mild, non-irritating diet may be given. The peculiarities of each individual case will have to be studied, and much will depend on the progress of the disease at what period of the treatment a more solid and a more nourishing diet may be given. The four different dietaries that Leube and Penzoldt have arranged (compare page 183) may form the bases of these dietary regulations. The first dietary should be allowed for at least ten days, then the second one should be given for a week, followed by the third one, etc. The severity of the symptoms in each individual case will determine at what period of the treatment one dietary is substituted for the other.

Cruveilhier, in his day, declared milk to be the most appropriate liquid food for ulcer cases. The composition of milk, in the first place, renders it very nourishing; in the second place, makes it non-irritating to the stomach. We see certain cases, it is true, in which the casein coagulates in the form of large lumps; if this is noticed, the administration of milk should be stopped. Reichmann has discovered that boiled milk leaves the stomach much more rapidly than unboiled milk, and that the lumps of casein that form when boiled milk is digested are much smaller than those formed when raw milk is digested. This point is of great practical importance. Leo and von Pfungen have also determined by a series of experiments that milk has certain antacid properties. Unfortunately certain persons cannot take milk in any form, not even if it is mixed with other substances or if correctives are added. It is bad practice, however, to refrain from administering milk, even if the patient claims that it does not agree with him. The attempt should always be made to give it in small quantities,—in teaspoonfuls,—or certain substances, like soda or lime-water, may be added. Occasionally so-called fat milk (Gärtner) can be borne even when ordinary milk does not agree with the patient. This preparation is made from cows' milk from which a certain proportion of the indigestible casein has been mechanically removed. Unfortunately the patients cannot be kept on a pure milk diet for a long period of time, because the demands of the body for food cannot be satisfied by milk alone. The administration of an exclusive milk diet is essentially a hunger cure, and causes the patient to lose considerable tissue.

The nutritive value of milk can be greatly increased by the addition of milk powder (100 gm. to 1 l.), and by the addition of one or several teaspoonfuls of condensed milk or of nutrose. Ewald advises the administration of flour soup boiled with milk. The advantage of this dish is that the casein coagulates in smaller flakes than in pure milk, and that it possesses greater nutritive value. In the majority of cases, particularly later in the treatment, finely divided amylaceous food,—tapioca, rice, maizena flour, Löffland's children's flour, and similar preparations,—may be added to the milk. In place of milk, buttermilk may be given; the latter contains very much less fat and sugar than pure milk, and is consequently less nourishing, but it is frequently relished by patients who cannot take pure milk. Of late years kefir has been administered with good results.

Sour milk, provided it does not contain too much acid, is also frequently relished. In southern Germany sour milk is a very popular article of diet. I know a number of physicians in this neighborhood who prescribe sour milk exclusively in ulcer, and claim to have seen uniformly good results from the administration of this food. Cream in small quantities is also often well borne.

Another article of diet that does not irritate the surface of the ulcer is meat jelly, as recommended by Fleiner. This food is prepared by boiling a chicken or some beef with calves' feet. Another similar preparation is the meat solution that Leube and Rosenthal describe. Leube recommends giving nothing during the first period of the treatment excepting milk, meat solution, and a little bouillon, with an egg or some barley. I am in the habit of limiting my ulcer patients in the beginning (provided I permit the administration of food by mouth) to an exclusively liquid diet, consisting of milk and bouillon with or without egg, possibly with the addition of somatose or nutrose. In the place of simple bouillon, gruel may be given later on. I do not, as a rule, allow meat solution for several days. Raw, beaten white of egg, and white of egg with water, may also be allowed. White of egg, like the casein of milk, seems to possess the power of combining acids, and even of neutralizing them (von Pfungen). If a diet of this kind is given, the patient is not well nourished; but this is not really necessary, for the chief object in view is to promote rapid healing of the ulcer.

I might briefly mention another proposition that has been made for feeding those patients who cannot tolerate milk, and who will not take it even in small quantities. Debove advises pouring the milk into the stomach through a soft stomach-tube. He found that his patients were able to digest milk that was introduced in this way, and did not vomit it. In order to increase the nutritive value of the milk he adds milk powder or condensed milk to ordinary milk, and evaporates it to one-half its volume.

Another appropriate article of food is sugar solution. According to the investigations carried out by Strauss[1] in my clinic, the secretion of acid in the stomach is smaller after the administration of a sugar solution than after the administration of a test-breakfast or after the introduction of carbohydrates in the form of undissolved starches. Sugar solutions, therefore, seem to be the appropriate article of diet, particularly in hyperacidity and in ulcer. They are, of course, contraindicated in all cases where there is a tendency to fermentation. Some 60 gm. of sugar in 20 per cent. solution may be given *pro die*. Milk-sugar is the least suitable one of the different varieties of sugar, because it possesses laxative properties. The best of all sugars is chemically pure dextrose; unfortunately this preparation is quite expensive. Ordinarily the commercial form of dextrose is good enough.

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[1] *Zeitschr. f. klin. Med.*, vol. xxix.

Recently a preparation has been introduced that is called casein nutrose. To judge from my personal experience with this product, it is very useful in ulcer cases, and promises to become a good substitute for meat; as it is, it improves the nutrition of the patients better than the ordinary substitutes in use. Nutrose is well absorbed, usually agrees with the patients even though they take it for a long time, and is even relished by some. It may be administered in large doses, 30 to 50 gm. a day, and can be given in all forms of soup, in milk, cocoa, etc. This preparation enables us to supply a large proportion of the albumin needed.

All the regulations that we have just enumerated constitute the first part of the cure, that should usually last about ten days. The patient during this time should be ordered to stay in bed. He should remain absolutely quiet, and should not even get up for the purpose of defecating or urinating. Warm Priessnitz compresses, poultices, or felt sponges dipped into hot water, should be applied to the region of the stomach. I usually employ warm Priessnitz compresses and use hot poultices in those case that suffer violent pain.

After some time, but not before ten days at least, the second dietary of Leube may be given. This consists of boiled calves' brains, boiled calves' thymus, boiled chicken, boiled pigeon, gruel, tapioca milk mush. Finely scraped raw beef may also be tried, as well as boiled calves' feet. Oatmeal is very nourishing, and may also be given at this stage.

After some time the third dietary may be given, consisting of scraped or half-rare broiled beefsteak, of scraped raw ham, mashed potatoes, cauliflower, and Zwieback.

The fourth dietary, that should be given as late as possible, offers much more variety. It consists of broiled chicken, pigeon, venison, prairie chicken, roast beef (rare), fillet of beef, and broiled veal (hind quarter); pike, shad, trout, rice, souffles, soups made of meat and flour, etc.

In regard to other articles of diet, the following may be said: Eggs do not agree with many patients; some authors, as Liebermeister, forbid them altogether for ulcer cases. Much will depend on the method of preparation; hard-boiled eggs frequently cause distress; hard-boiled eggs, however, mashed fine, are usually much better borne. The best form in which to give eggs is as white of egg only slightly coagulated.

Everything that can irritate the stomach mechanically, as leguminose vegetables, black bread, crusts of bread, skin, tendons, fruit pips, and stones of berries, etc., should all be strictly avoided for a long time. Even after all symptoms have disappeared, and we are justified in assuming that the ulcer is healed, the diet should be carefully supervised for a long time.

Alcoholic beverages should be altogether interdicted, particularly in the beginning of the cure. Only if there is great weakness may wine-soups or red wine mixed with an alkaline water be permitted. If the patients are weak, enemata of wine together with peptone may be found useful.

Ewald recommends Ross's "Kraft beer" in particular. He gives it with Biliner or Ems water. These waters are permitted to stand for some time in order to allow the escape of a large proportion of the carbonic acid gas. This beverage, however, does not possess great nutritive value. Coffee should never be allowed in ulcer; tea may be given.

The second part of the treatment consists in the administration of alkaline saline mineral waters or of their salts. These waters have been popular for a long time in the treatment of all diseases of the stomach, particularly of ulcer. Of these, Carlsbad water is the most popular. All that we know in regard to the favorable effect of this water is empiric, and so far no satisfactory explanation of its effect has been forthcoming.

The main ingredients of Carlsbad water are chlorid of sodium and carbonate and sulphate of sodium. Ziemssen attributes the main effect of Carlsbad salt to the Glauber salt that it contains. Leube is inclined to attribute the good effect of Carlsbad salt and water to the power of these preparations to cause evacuation of the stomach-contents; at the same time he argues that the sodium chlorid exercises a favorable effect on digestion, and that the carbonate of sodium dissolves the mucus and reduces the degree of acidity of the stomach-contents. The latter two properties of the water he considers of subordinate importance.

From a theoretic point of view this explanation is not altogether satisfactory. The stomach-contents are probably not evacuated more rapidly if the water is given in the customary way, for, as a rule, the water is prescribed in doses of about one-quarter to one-half a liter, or the salt in doses of one or two teaspoonfuls, early in the morning before breakfast. In cases of ulcer, however, the stomach is always empty in the morning, provided there are no complications. The Carlsbad preparations cannot possibly exercise an antacid effect, for the reason that the stomach in the morning before breakfast contains no acid gastric juice. Of course, if ectasy or atony is present, this is different, but we are not dealing with such cases in this place. At the same time it cannot be denied that Carlsbad water exercises a certain influence on the secretion of gastric juice. Thus, Jaworski found that large quantities of Carlsbad salt or water inhibit the secretion of gastric juice and impede its digestive powers for several hours. He also showed that medium and large quantities given for a long time produced a progressive decrease in the secretory powers of the gastric mucosa.

The results obtained by Spitzer, who studied 18 cases of stomach disease during a four weeks' course in Carlsbad, are also interesting. He found that in the course of this treatment the motor power of the stomach was increased in the majority of cases and damaged in none of them; that the superacidity of the stomach-contents soon disappeared, and that normal values were obtained; further, that subacidity was never produced. This last result is particularly important for the question we are discussing.

Another important action of Carlsbad water is the regulation of the bowels. In addition, the relatively large quantities of warm water that are taken exercise a beneficial effect in two ways—first, they quiet the stomach; second, they cleanse it thoroughly and stimulate the activity of the intestine. From a practical point of view, therefore, we may consider the value of Carlsbad water established. The exact way in which it acts, however, is not yet satisfactorily explained.

If Carlsbad water cannot produce a sufficient evacuation of the bowel-contents, the dose must either be increased or from one-half to one teaspoonful, or even more, of artificial Carlsbad salt added. Of course, natural or artificial Carlsbad salt may be used throughout instead of the Carlsbad water; one to two heaping teaspoonfuls of the salt are dissolved in one-quarter to one-half a liter of lukewarm water, and the whole taken in the course of about half an hour. The patient should wait at least half an hour after taking the water before he eats his breakfast. In the majority of patients this dose is sufficient to produce one or two evacuations of the bowels. If the action is too severe, the quantity must be reduced; if not sufficient, increased.

Many authors, like Ziemssen, prefer the natural or the artificial salt to the water. The chief advantage of giving the salt is that the concentration of the solution can be regulated to suit the peculiarities of each individual case. If the stools are not regulated by the administration of Carlsbad salt, other laxatives should not be given; the bowel-contents should be removed by irrigation.

These are the essential features of a "rest-cure." In cases of ulcer that are not very old, the methods that we have described, as a rule, suffice, provided they are applied for a sufficiently long time.

A brief summary of this method of treatment reads as follows: The patient should be instructed to remain in bed during the first period of the course—at least, for the first two weeks. During this time he should be kept as quiet as possible. Priessnitz compresses, or hot poultices if there is much pain, should be applied to the region of the stomach and frequently renewed. The ice-bag should be applied only in those cases where symptoms of impending hemorrhage are observed or where a hemorrhage has just occurred.

During the first period of this cure the patient should not be allowed to leave the bed under any circumstances, not even for the purpose of urinating and defecating. The contents of the bladder and the bowels should be deposited while the patient is lying on his back. In the morning, before breakfast, the patient should take Carlsbad salt,—one or two teaspoonfuls dissolved in about one-quarter of a liter of warm water or of Carlsbad water. If necessary the dose may be increased.

In the beginning the diet should consist exclusively of milk and bouillon, with or without an egg, possibly with the addition of a little somatose or, better, nutrose. If milk cannot be borne in any form or if it agrees only when taken in very small quantities, meat solution, about 100 gm. pro die, or meat-jelly, may be given. All liquid articles of food should be taken lukewarm. If milk is well borne, two liters may be given a day, but only in divided doses, so that small quantities are given at each meal. An increase of this quantity should be gradual. The longer the patient can adhere to this strict diet the better.

If treatment is begun immediately after a gastric hemorrhage, the administration of food by mouth should be stopped altogether and the patient fed exclusively by rectum for the first three or four days or still longer. I refer to the section on Hemorrhage for the details of the treatment of this condition.

[Opinion is divided as to whether rectal alimentation increases or diminishes gastric secretion. Following the observations of Winternitz, it was believed that the gastric acidity was increased by nutrient enemata, but the studies of Ziarko[1] go to show that the acidity of the gastric juice is really decreased by rectal feeding. Bourget, on the other hand,[2] discards rectal feeding, believing that food thus introduced is but slightly absorbed, and that by reflex action it increases gastric secretion.—Ed.]

If the ulcer is not very old, vomiting and pain usually disappear within a few days under this treatment, so that, as a rule, it is unnecessary to administer narcotics or other drugs.

If the pain and all other distress disappear after about ten days of this treatment,—and this is the rule,—the administration of Carlsbad salt should, nevertheless, be continued for some time. The diet, however, may be more liberal; a number of softened cakes or softened Zwieback may be given, or tapioca or rice boiled in bouillon or milk, leguminous soup, etc. After a few days very digestible meat dishes may be allowed: calves' thymus boiled in bouillon, boiled pigeon, or, later, raw and scraped ham. It is best to wait for a time before allowing beefsteak; when this is given, it should be scraped and quickly roasted in butter with a little salt without the addition of any other condiment. Potatoes should be given only mashed; the same applies to carrots. At this period boiled chicken may also be eaten. Roast chicken should not be given until later.

If it is at all possible the patient, as we have said, should spend the first two weeks in bed. At the expiration of this time he may be allowed to get up for a short time each day, provided he is free from pain. Gradually he may be allowed to sit up for a longer time. The cure usually consumes from four to six weeks, provided there are no complications or complications do not arise during this time. Even after the cure proper is over the patient should be very careful for a long time as to what he eats, and should avoid all irritating articles of food, all condiments or mechanically irritating articles of diet, all hot and very cold beverages, fruit-ices, alcoholic beverages, etc. Unless he does this, recurrences are apt to take place. During the cure small meals should be eaten; after the cure is over the patient may eat more at each time and can allow longer intervals to elapse between meals.

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[1] *Prager med. Wochenschr.*, 1899, No. 14.

[2] *Therap. Monatsh.*, July, 1900.

It will hardly ever be necessary to send patients to Carlsbad, Neuenahr, Vichy, or similar watering-places for the sake of undergoing a cure. It is better to advise a vacation to some mountain resort, where the air is invigorating and pure. The diet, of course, should be carefully regulated during this time. In order to improve the general nutrition of the patient during this stage iron preparations and arsenic may be given. It is best, however, not to administer these drugs too early. I have had good results with liq. ferri album (Drees). Le Gempt[1] reports the same. There are, however, many other preparations that are just as good. Ewald has described a very suitable and at the same time cheap method of giving iron; he prescribes a 2 to 3 per cent. solution of the sesquichlorid of iron, and gives a teaspoonful three times a day in a wineglassful of egg-water (one part white of egg, two parts of water). He advises his patients to take this remedy through a glass tube in order to protect the teeth. Arsenic may be given together with iron in order to stimulate the nervous system and to improve its tone. Liebreich advises giving it in the form of arsenious acid, and not as Fowler's solution. If possible, iron and arsenic should be given only in liquid form. The arsenic and iron waters of Levico, Roncegno, and other places, may be given in place of iron and arsenic preparations. Ewald and Dronke have performed a number of metabolic investigations with these waters, and have shown that they are very effective; the dose should gradually be increased. It is well to interrupt the treatment for a short time occasionally. If any untoward symptoms appear, these two drugs should, of course, be stopped at once.

[Fuetterer,[2] believing that the resistance to healing observed in gastric ulcer is largely dependent upon the decrease of hemoglobin in the blood, recommends a daily dietary of freshly expressed juice from five pounds of beef.—Ed.]

This treatment leads to the best results. There are exceptional cases, of course, in which nothing is accomplished by a rest-cure and in which vomiting persists notwithstanding. In these cases it is best to proceed in the same manner as in hemorrhage from the stomach—namely, to place the organ at rest for a time and to feed the patient exclusively by the rectum. Bouveret recommends feeding these patients through a sound. It is not necessary to introduce the sound into the stomach; all that is needed is to insert it as far as the middle or lower third of the esophagus. Bouveret claims to have seen good results from this method of feeding in many rebellious forms in which vomiting was obstinate. In other cases the reason why a rest-cure fails is that complications exist.

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[1] *Berlin. klin. Wochenschr.*, 1886.

[2] *Jour. Amer. Med. Assoc.*, January 11, 1902.

This "rest-cure" that we have just described, and that, of course, must be modified to suit the peculiarities of each individual case, is undoubtedly the most reliable method and the one that has stood the test of practical experience. This treatment can be recommended both for severe forms of ulcer and for mild and recent forms. In the latter the beneficial results are obtained much more rapidly and can be predicted with greater certainty.

There are a number of cases, however, in which this rest-cure cannot be carried out. This may be due to a variety of external circumstances or to the fact that the patients cannot make up their minds to undergo the treatment.

Many different authors, therefore, have attempted to replace this method of treatment by others, and of recent years the medicamentous treatment of ulcer has become quite popular. There can be no doubt, however, that the dietetic rest-cure is the most rational method of procedure. Many do not like to employ this treatment, because they claim that it is so difficult to carry out. I do not consider this objection valid in view of the positive results that invariably follow its employment.

An ulcer may, of course, occasionally heal without any treatment. Many ulcers, as we know, run an altogether latent course, and the only evidence we may have that an ulcer existed during life may be a scar that we find post mortem. But this can hardly justify the ambulatory treatment of cases of ulcer nor make this method of treatment rational. Anyone who is afflicted with ulcer of the stomach should remain in bed, and should not attempt to follow his usual occupation. A physician should not be influenced by the objections that the patients offer; very few patients will refuse to follow the directions of a physician if they can be taught what the physician is attempting to do and what the dangers of their condition are. In many instances drugs can aid the treatment,—can relieve certain symptoms,—but they are indicated only when employed together with one of the above-named methods with the dietary rest-cure. Drugs alone can accomplish very much less than if they are employed together with a rest-cure; I am of the opinion that they should be used only in emergencies.

Various remedies have been advised in place of a rest-cure. The chief of these are the preparations of bismuth. As early as 1786—that is, more than one hundred years ago—Odier recommended bismuth preparations as a panacea for spasm of the stomach. In the first half of the last century subnitrate of bismuth was the most popular remedy in ulcer. This drug was employed for a long time; some authors recommended it; others condemned it. Of recent years Fleiner, following Kussmaul's advice, again called attention to subnitrate of bismuth; he advises giving this preparation in the same large doses that the older physicians employed. Nowadays 15 gm. are recommended, and long ago a number of physicians and clinicians, particularly Frenchmen, recommended similar doses and employed them. Leube calls attention to the fact, however, that the remedy was rarely given alone, but usually with morphin, opium, etc.

Kussmaul and Fleiner employed the remedy chiefly in order to protect the injured portion of the stomach from further injury and to prevent the gastric juice and irritating food from touching it; in other words, they used it as a protective measure. Fleiner and Kussmaul advised giving it for this purpose as follows:

The patient's stomach is washed out early in the morning before breakfast. This is done in order to cleanse the stomach as completely as possible. As soon as the wash-water runs clear, from 10 to 20 gm. of subnitrate of bismuth are suspended in 200 c.c. of lukewarm water, the suspension immediately poured into the funnel, and washed down with 50 c.c. of water. The patient is instructed to lie in such a position that the ulcer comes in immediate contact with the suspension of bismuth. At the expiration of a few minutes—five to ten—the bismuth is so completely precipitated that the water in which it was suspended can be allowed to flow from the stomach and the sound removed. The patient is ordered to remain in the same position for half an hour and then to eat his breakfast. This bismuth treatment should be employed daily in the beginning; later every other day or every third day. It should be continued as long as it seems necessary. Fleiner claims never to have seen any symptoms of poisoning even after using from 300 to 400 gm. of bismuth. In those cases in which the passage of the stomach-sound is contraindicated Fleiner advises drinking 10 gm. of subnitrate of bismuth suspended in a glass of lukewarm water early in the morning before breakfast. Boas prefers the carbonate of bismuth to the subnitrate, chiefly because the former salt interferes less with the function of the intestine. Fleiner explains the action of bismuth by assuming that the precipitate of bismuth protects the diseased portion of the mucosa, and in this way prevents the gastric juice from exercising its eroding and irritating effect on the sensory nerve-endings that are exposed in the ulcer. The favorable results of Fleiner have been corroborated by many other authors,—for instance, Fischer, Crämer, Savelieff, Witthauer, and others.

[A method similar to that of Fleiner has recently been advocated by C. Pariser,[1] who advises that there should be taken from 15 to 20 gm. of bismuth subnitrate stirred in water, on an empty stomach, in the morning, this to be followed by a little pure water. The patient is then directed to lie quietly on the back for three-quarters of an hour, after which he is allowed to take coffee and a roll. Pariser states that he has never seen any toxic effect from large doses of bismuth, but, owing to its expense, he has lately replaced it by a mixture of 60 gm. each of chalk and talcum, to which are added 15 gm. of magnesia usta. He sees advantage in this latter mixture in the laxative and antacid effects of the drugs, and also in the fact that they do not blacken the stools, thereby concealing slight hemorrhages that might otherwise pass unobserved.—Ed.]

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[1] *Deutsch. med. Wochenschr.*, April 10, 1902.

Matthes has performed a number of animal experiments that are of particular interest. In the first place, he discovered that bismuth introduced into the stomach settled at the lowest portion of the organ within ten or twenty minutes after its administration. He determined this by giving the preparation to dogs and then performing gastrotomy at the expiration of this time. If he allowed the animals to live for several hours, he found that the bismuth was uniformly distributed over the whole internal surface of the stomach. Matthes, therefore, considers Fleiner's direction that the patient should remain in a definite position for some time after the administration of bismuth superfluous.

If an artificial solution of continuity of the gastric mucosa is produced and bismuth administered for some time, a thick crust of bismuth will be found adherent to the wound ; it seems, therefore, that this drug has the property of forming a covering like a powder-bandage. Matthes was also able to corroborate Fleiner's statement that the administration of bismuth stopped the pain. Savelieff also saw favorable results throughout. In opposition to Matthes, however, he attaches great significance to the position of the patient and the length of time during which he should remain in this position. The latter author regards bismuth as the sovereign remedy in cases that are up and about. In recent cases he also recommends a rest-cure and a milk-diet, and states that these methods are the most effective ones.

Crämer, who advises his patients to drink a suspension of bismuth and water in the morning before breakfast, also reports favorable results. Witthauer does the same. Rosenheim's experience corresponds with that of all the other authors. Boas, on the other hand, reports less favorable results ; in some cases, failures.

My own experience extends only to cases that were treated in the clinic. I have never limited myself to the administration of bismuth, but have always insisted on a rest-cure and a careful regulation of the diet at the same time. I never order bismuth suspension to be introduced into the stomach by the sound, but I simply direct the patients to drink the suspension. As a rule, the patients took the remedy in one dose of 10 gm. some time in the course of the forenoon,—that is, at a time when the stomach was empty. I can corroborate the statement of most of the above-named authors that the effect of this remedy is very favorable. There can be no doubt that it relieves the pain considerably. I, therefore, feel justified in recommending bismuth as a valuable adjuvant to a dietetic rest-cure. I advise its administration not only in old cases, but in all cases of ulcer.

Another drug that has frequently been recommended and has at other times been condemned is nitrate of silver. This drug has been employed for a long time in the treatment of ulcer. Johnson was the first to recommend it. He used it in the treatment of epilepsy, and found that all gastric symptoms that were present in his cases disappeared after its administration. Of late years Gerhardt, in particular, has warmly recommended the administration of silver nitrate in ulcer. He claims to have seen dozens of cases in which all gastric symptoms caused by ulcer disappeared immediately after the administration of silver nitrate, and, on the other hand, to have seen many cases in which it exercised no effect whatever, and even increased the distress of the patient. According to Gerhardt, nitrate of silver acts well, particularly in those cases that suffer from severe pain when the stomach is empty. He thinks that in these instances it acts as an antacid. If silver nitrate solution is added to stomach-contents that react to Congo-paper (for instance, from a case of pyloric stenosis) until no precipitate is formed, the reactions for hydrochloric acid will not be obtained (Gerhardt); the silver is precipitated as silver chlorid, and the nitric acid combines with the albumin to form an insoluble precipitate. Gerhardt argues that moderate quantities of stomach-contents may be neutralized by silver nitrate. He administers one or several centigrams of silver nitrate in solution once to three times daily when the stomach is empty; at the same time he instructs his patient to occupy a position that will cause the remedy to come in contact with the ulcer.

Silver nitrate, therefore, sometimes exercises beneficial effects. We are not justified in doubting this if we remember what Gerhardt reports; at the same time the antacid action of silver nitrate must be minimal when we consider what small quantities of the drug are introduced into the stomach. This can easily be demonstrated by mixing a stated quantity of gastric juice with a 0.2 to 0.3 per cent. solution of silver nitrate and by determining the residual acid by titration. We have done this in a number of experiments. The antacid action of nitrate of silver is probably still less if the drug is taken on an empty stomach—that is, at a time when in simple uncomplicated ulcer no gastric juice containing hydrochloric acid is, as a rule, present. I do not think, therefore, that this is an altogether satisfactory explanation of the action of silver nitrate, nor do I believe that we have as yet determined the exact conditions under which it is effective or useless. At the same time the fact that such experienced observers as Gerhardt and Boas speak in favor of this remedy should encourage us to further investigation.

Boas recommends the silver nitrate treatment, particularly in mild cases of ulcer and in follicular ulcers; also in patients that cannot undergo a typical ulcer cure. He begins the treatment by giving solutions of 0.2 to 120 in tablespoonful doses three times a day, and always on an empty stomach; then he increases the drug to 0.3 to 0.4. He states that a careful regulation of the diet should be insisted upon. The disadvantages of this drug are its bad taste and the occasional occurrence of nausea after its administration. Rosenheim recommends silver nitrate, particularly in those cases in which there is hyperesthesia of the stomach and in which the organ seems particularly intolerant to food.

Another medicamentous method of treating ulcer of the stomach may be briefly mentioned in this place—that is, the treatment with chloroform, a procedure that has recently been warmly advocated by Stepp. He has reported a number of cases that seem to indicate that the remedy can exercise a favorable effect on the course of ulcer. Stepp, however, makes one statement with which I hope the majority of his readers will not agree—namely, that the rest-cure that Leube and Ziemssen recommend cannot possibly be carried out in private practice. I believe that the rest-cure can be carried out in all cases, provided the physician in attendance demands it with sufficient energy and sees that it is carried out correctly. Stepp considers the most important part of Leube's cure—that is, the dietetic part—essential. He, too, like every other physician nowadays, attaches particular importance to the regulation of the diet. In the beginning he advises a milk diet; in the second week, meat broth; later, with the addition of one or two eggs, and still later, easily digestible meat dishes. We see, therefore, that Stepp accepts the most important part of Leube's cure. I fail to see why patients who are living on a diet that in no way satisfies the caloric demands of a workingman should not remain in bed during the first two weeks.

Stepp administers chloroform together with bismuth; chloroform 1.0, aq. dest. 150.0, bismuth. subnit. 3.0, one to two teaspoonfuls every hour.

Another remedy that may be mentioned is condurango bark. Gerhardt states that this drug can frequently heal gastric ulcer if the diet is suitably selected, and that it is particularly efficient in old gastric ulcers in emaciated subjects. My experience has taught me that condurango bark, which Friedreich, as we know, regards as a specific for carcinoma, acts only as a stomachic.

In addition I might refer to iodid of potassium that Ord has recommended, and that this author administered together with carbonate of sodium. Still another drug is the sulphate of atropin, which Wojonowitsch recommends. This investigator claims that it reduces the secretion of gastric juice.

Whereas all the methods that we have described are intended to exercise a direct influence on the ulcer, alkalis are used only symptomatically—that is, in order to combat the hyperacidity and to relieve the attacks of pain. Ewald advises neutralizing the hyperacidity of the gastric juice by alkalis combined with rhubarb and cane- or milk-sugar. This mixture he gives every hour in small doses. Bouveret administers from 8 to 14 gm. of bicarbonate of sodium a day. I am in the habit of giving alkaline remedies only during meals or immediately after meals—that is, at the time of hyperacidity. In pure uncomplicated ulcer hyperacidity is periodic—that is, it appears as soon as the gastric mucosa is irritated by the ingesta; all that is needed, therefore, is to administer some alkali after each meal at the height of the production of acid or immediately before. In those cases that I have called continuous secretion of gastric juice, and that are quite frequently combined with ulcer, we must proceed differently. Here it seems rational to administer alkali at more frequent intervals between meals—in some instances even immediately before eating. The time at which the alkaline remedy is to be administered is determined by the nature of the perversion of the gastric secretion. In uncomplicated ulcer, it appears to me, alkalis should be administered about one hour after meals, and if the meal is small, still sooner afterward. I usually employ the following prescription : Bicarbonate of sodium, magnesia usta, of each, 10.0 ; sugar of milk 15.0 ; and add a little rhubarb if it is desired to stimulate the function of the intestine. The patients receive this powder in bulk from the apothecary, and I usually order them to take half a teaspoonful or a little more of the powder immediately after eating. If the pain does not stop altogether, I sometimes add a little extract of belladonna. As a rule, the administration of other narcotics is unnecessary if the dietary rest-cure is carried out conscientiously. In almost all instances pain disappears within a few days after the treatment is begun.

If cardialgic attacks occur, nevertheless,—and this will be rare,—narcotics may be exhibited. The most important of these is morphin, which exercises a distinct inhibitory influence on the secretion of hydrochloric acid. This has been found to be the case by Leubuscher and Schäfer, and independently of these by Hitzig.

[According to the later publication of Riegel's experiments on the effect of morphin upon the secretion of gastric juice,[1] he was led to another conclusion ; for in this article he says that, contrary to general belief, morphin increases, rather than decreases, the gastric secretion. Atropin, on the other hand, diminishes the secretion and also inhibits motility and cramp. These conclusions were substantiated by the experiments on animals made by Alder,[2] and by the still later observations of Riegel.[3]

Hirsch[4] found that, by the subcutaneous injection of morphin in dogs, there resulted at first a decrease, followed by a marked increase in the secretion of HCl. The influence of the drug increased progressively with the increase of the dose, and the hypodermic effect was more marked than when the drug was taken by the mouth. He also found that there was an increase of the intragastric pressure, apparently from contraction at the pylorus, accompanied by increased peristalsis and an increased flow of gastric juice. From atropin he found that the HCl was decreased, and that it reappeared and was again normal after six hours.

Riegel reports that pilocarpin stimulates the flow of gastric juice ; on the other hand, Schiff,[5] from his experiments, concludes that HCl cannot be increased by pilocarpin, but that the watery secretion containing pepsin is readily excited. He agrees that HCl secretion is markedly diminished by the effect of atropin. It may be concluded that morphin increases the secretion of HCl, that atropin diminishes it, and that pilocarpin increases the flow of gastric juice.—Ed.]

According to Gerhardt, atropin is more efficient in many cases than morphin. Sée praises strontium bromid in hyperchlorhydria. Other remedies that are recommended for this condition are the extract of cannabis indica, which Sée also recommends; then different preparations of codein, particularly the phosphate of codein, which Ewald and Boas recommend. The last-named remedies have this advantage over morphin, that they interfere less with the peristaltic movements of the intestine. The preparation of hemp has the disadvantage of frequently causing disagreeable states of psychic excitement without, at the same time, exercising a distinct analgesic action. I might mention incidentally that leeches applied to the region of the stomach have also been recommended for violent cardialgic attacks. Some authors use mustard-plasters or blisters. I think that the last-named methods can be dispensed with.

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[1] *Zeitschr. f. Aerzte*, 1898, 17.
[2] *Zeitschr. f. klin. Med.*, vol. xl., p. 248.
[3] *Ibid.*, p. 317.
[4] *Centralbl. f. innere Med.*, January 12, 1901.
[5] *Arch. f. Verdauungskrankh.*, vol. vi., p. 107.

In those cases where the pain is limited to a small circumscribed portion of the anterior wall, and in which circumscribed peritonitis is suspected, it is always best to apply a small ice-bag that, in order to avoid pressure on the abdomen, may be suspended from a hoop attached to the bed.

A number of remedies have been recommended for the vomiting that occurs in ulcer. As a rule, we can get along very well without them. If the rest-cure is correctly carried out; if the diet is regulated; and if alkalis are administered when necessary, vomiting almost always stops. I have never found it necessary to employ special remedies for it.

The vomiting of blood is a much more important symptom, and usually calls for energetic interference. The patient, of course, should be advised to remain perfectly still in bed and to avoid all movements. The stools and the urine should be passed while the patient is lying down. A small ice-bag is applied to the stomach region, and no food is given by the mouth. The thirst is relieved by washing out the mouth or by giving the patient small pieces of ice that he should be instructed to allow to melt on the tongue, for swallowing large pieces of ice may be dangerous. If the hemorrhage is very severe, subcutaneous injections of ergotin (ergotini 2.5, aq. dest., glycerini, āā 5.0) should be given; and, if necessary, repeated. If the patients are very much excited, small injections of morphin may be given in order to quiet them. In place of ergotin the fluid extract of hydrastis canadensis or of hamamelis virginica, or of chininum ferro hydrochloricum, may be administered; the former in drop or teaspoonful doses several times a day, the latter in powder-form or in solution in doses of 0.5 to 1.0. Bismuth is also recommended as a hemostatic. The acetate of lead or the chlorid of iron may be given to stop bleeding. If possible it is best, however, to avoid giving any of these drugs, for the best treatment is to keep the stomach completely at rest.

[While almost all remedies introduced into the stomach for the relief of gastrorrhagia are of questionable utility, even when not directly harmful, there is reasonable ground for hope that a solution of the new drug, "adrenalin chlorid," may prove to be actually beneficial in certain cases of severe hemorrhage from peptic ulcer. Relief seems to follow the administration of a solution of 1 : 1000, in doses of 10 to 30 drops, to be repeated as circumstances warrant. It is best given in a spoonful of water immediately after vomiting. As to the subcutaneous employment of the drug, it is probably wiser to wait for larger experience before deciding on its usefulness, but it promises to be more valuable than ergot.—Ed.]

A more rational method of procedure in emergencies is washing the stomach with iced water. Ewald reports excellent results from this procedure in two cases. Wiel has recommended washing out the stomach with hot water (42° C.), and other authors advise drinking water as hot as it can be swallowed.

If the patient is not very much weakened from the hemorrhage, no food at all should be given in the beginning, not even by rectum. If the patient, however, is much weakened and the pulse is small, nutritive enemata, preferably altogether liquid, should be administered. I refer to the general part of this work (p. 209) for the methods of preparing these rectal enemata.

If the hemorrhage is profuse and if symptoms of severe weakness of the heart develop, with a thready, very rapid pulse, coolness of the extremities, a drawn facial expression, stupor, and other symptoms of collapse, stimulants should be given. The best of these is camphor given hypodermically, either as camphor or camphor-ether, at frequent intervals. Enemata of wine bouillon may also be given. Another method that is efficient in advanced anemia and weakness of the heart is so-called autotransfusion. This consists in bandaging the extremities tightly, so that more blood is forced into the internal organs. If the power of the heart does not increase despite all these measures, and if the pulse grows weaker and finally can no longer be felt, infusions of salt should be tried. I have seen remarkable results from such salt infusions in a number of cases that were in a stage of advanced collapse. The method is very easy, and the adjuvants and apparatus that are necessary can be procured anywhere. All that is needed is a so-called physiologic (0.6 per cent.) salt solution, heated to about 37° C. (98.6° F.), a cannula of a slightly larger caliber than the cannula of a Pravatz needle, a rubber tube, and an irrigator. In emergencies a simple glass funnel may be employed. The salt solution is poured into the glass irrigator or the funnel, and the tube and cannula filled with fluid, and the cannula then inserted into the subcutaneous cellular tissues. An assistant or attendant is instructed to elevate the glass funnel slightly while the physician holds the cannula in place and regulates the flow of water by pressing the tube. I have been in the habit of performing the injection in the anterior part of the thorax underneath the clavicle. Rosenheim recommends injecting the salt solution underneath the angle of the scapula. If the skin is massaged around the point of injection, 500 c.c. or even more can easily be injected.[1] If necessary, several injections can, of course, be made in different parts of the body.

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[1] If, in exceptional cases, the panniculus adiposus is so tense that infusion cannot be performed in the manner described, the salt solution may be forced into the cellular tissues by an ordinary syringe with a plunger.

[It may not be necessary to call attention to the author's implied caution against the premature employment of injections of normal salt solution in case of hematemesis. Nevertheless, the practice has become so common that the caution is emphasized. I have several times seen hemorrhage apparently re-excited by means of enteroclysis and hypodermoclysis when the patient's condition did not demand a further distention of the blood-vessels. In this connection it may be pointed out that a similar danger lies in the use of stimulants with the intention of increasing blood pressure, when, in fact, the low vascular tension which is sought to be overcome, if not allowed to become very extreme, is an element of safety to the patient.—Ed.]

Subcutaneous injections of blood, which Ziemssen recommends, may also be given in place of salt-water infusions. The chief difficulty here is that frequently the blood can not be procured at once; the procedure, moreover, is rather difficult. In general it will rarely happen that hematemesis leads to such severe symptoms of collapse that the last-named measures—infusion, transfusion, etc.—have to be employed.

The same method that we have described in the treatment of hematemesis may also be called for in cases in which no blood leaves the body after the hemorrhage, for quite frequently the blood that is poured into the stomach during the gastric hemorrhage is not vomited, but passes into the intestine.

If the hemorrhage has stopped for several days, the administration of food by mouth may be slowly begun. I need hardly emphasize that nothing but cool liquid food should be given, and this only in small quantities. The most suitable article of diet is milk, which should be given in teaspoonful doses in the beginning. Later bouillon may be given, and still later bouillon with egg or nutrose, pultaceous soups, and gradually soups with other additions.

The chief indications for operative interference in ulcer of the stomach are, in the first place, hemorrhages. If there is a single severe hemorrhage, it will, as a rule, be impossible to perform so serious an operation rapidly enough to control the hemorrhage in time; more frequently we will be called upon to treat the severe anemia that follows hemorrhage. This condition can be treated by the above-named methods of infusion. If, however, the patient suffers repeated hemorrhages, so that he becomes very much exhausted and his life is in danger, operative interference may be indicated. In these instances a fistula may be made between the stomach and the small intestine,—that is, a gastro-enterostomy,—provided the ulcer cannot be found, cauterized, or excised. As soon as gastro-enterostomy is performed the irritation is removed and there is no further cause for hemorrhage. Even in very profuse hemorrhages that follow one another in rapid succession an operation may be indicated.

Another complication that, on the whole, is rare, but that calls for energetic treatment, is perforation. Spontaneous healing of this lesion can occur only if the stomach is completely empty when perforation occurs. In this instance a circumscribed peritonitis develops that may ultimately lead to the formation of adhesions. If, however, perforation occurs when the stomach is full; if stomach-contents enter the abdominal cavity—the only possible way of saving the patient is to operate. The operation, however, should be performed as soon as possible after the perforation occurs—that is, as soon as the first shock is over. The later we operate, the smaller the chances of recovery. It is important to know that the favorite location of perforation is the anterior wall of the stomach, in particular the cardiac portion of the organ, for here the conditions that favor the formation of adhesions are particularly poor. We should refrain from operating only in those cases in which we are positive that the stomach was completely empty when perforation occurred; if we do not know this, we should certainly operate. I need hardly mention that in those cases in which no gastric contents have entered the peritoneal cavity no food should be given by mouth for some time; also that everything should be done to put the stomach and intestine at rest—that is, ice compresses and opium should be employed (the latter, of course, only by the rectum as a clyster or in suppositories). Wherever there is even the slightest suspicion of a perforation, the administration of minimal quantities of food by mouth is contraindicated for several days. We should be particularly careful in this respect, as the symptoms of collapse that immediately follow perforation may disappear in many cases, so that the patients seem to recover after a short time. A number of cases are reported in the recent literature that point out this mistake, for the rapid disappearance of the symptoms of collapse has led a number of investigators to doubt the diagnosis of perforating ulcer that they originally made, and to give small quantities of food; in several instances the complete picture of fatal peritonitis from perforation developed a short time after.

Another indication for operative interference is the persistence of cardialgia and vomiting, even though a strict rest-cure is carried out. The patients in these instances continue to lose flesh and to decline. As a rule, however, these are not cases of uncomplicated ulcer, but cases in which the sequelæ of ulcer or complications have developed.

The most important indications, therefore, for surgical interference are perforation and repeated gastric hemorrhages that endanger the life of the patient. The latter indication is probably rarely present; in most instances the hemorrhage will be so severe that an operation cannot be performed in time. In these cases in which it was possible to operate, the ulcer has been either excised or cauterized, or a gastroenterostomy has been performed. Surgeons frequently encounter the difficulty of not being able to find the ulcer even after opening the stomach. It is an easy matter to find old ulcers of the stomach, as the neighboring parts are always indurated and there is usually stenosis of the pylorus; it is a difficult matter, however, if the ulcers are small and are situated in the anterior or posterior wall of the stomach. A number of cases are on record in which the ulcer was not found (Eiselsberg,[1] Hirsch,[2] and others).

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[1] *Arch. f. klin. Chir.*, vol. xxxiv.

[2] *Berlin. klin. Wochenschr.*, 1896, No. 38.

It has been questioned whether or not in future endoscopy or gastroscopy will render the discovery of the ulcer easier; in general it may be said that ulcers of the anterior wall are better removed by excision, those of the posterior wall and the pylorus, particularly if adhesions have formed, by cauterization.

[As bearing upon the probable value of surgical intervention may be cited the conclusions reached by Greenough and Joslin,[1] based on the study of 187 consecutive cases of gastric ulcer occurring in the Massachusetts General Hospital between the years 1888 and 1898. They show that the disease is more frequent in Boston than in many other large American cities, and that it is five times more common in women than in men. The average age in men is thirty-seven years; in women, twenty-seven. Hemorrhage was present in 81 per cent. of these cases, and caused death in 17 per cent. of the male patients and in only 1.27 per cent. of females. In 70.1 per cent. the patients were pale and the anemia was found to be of the chlorotic type. Perforation occurred in 3.2 per cent. of cases, none of which recovered. Of 114 cases, 80 per cent. were discharged cured or relieved, but at the end of an average period of five years only 40 per cent. remained well. The mortality at the same time (due to gastric disease) was 20 per cent.; among males it was 30 per cent., and among females 9 per cent. With these points in mind the observers concluded that a mortality of 8 per cent. and the failure of medical treatment to effect a lasting cure in 60 per cent. indicate the need of surgical intervention in other than emergency cases of this disease.

In a recent article Fleiner[2] reviews his treatment of gastric ulcer, stating that in the course of ten years he has treated over 300 cases without a single death, without a single hemorrhage, and without uncontrollable vomiting. In 27 cases it was necessary to resort to surgical intervention for the purpose of facilitating the emptying of the stomach. He states that resection of the ulcer, although theoretically promising, does not appear to him to be so satisfactory as expected. He attributes the greatest importance to dietetics and absolute quiet in bed, and he insists upon the value of subnitrate of bismuth as above described, pointing out as its chief disadvantages that it tends to induce constipation, that it occasionally gives rise to the formation of concretions; he doubts the correctness of the statement that it has in some instances a poisonous action.—Ed.]

Occasionally cicatricial stenosis of the pylorus that may be due to an ulcer calls for surgical interference. I refer to the sections on Stenosis of the Pylorus and Ectasy for the different operations that can be performed for this purpose.

Adhesions and perigastric inflammation that follow ulcer may occasionally call for operative treatment if they cause severe symptoms; the same applies to subphrenic abscesses. I refer to surgical hand-books for the details of all these operations.

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[1] *Amer. Jour. Med. Sci.*, August, 1899.

[2] *Münch. med. Wochenschr.*, June 3 and 17, 1902.

A few remarks may be permitted on the employment of mineral waters. We have already mentioned that Carlsbad water and similar springs enjoy a great reputation in the treatment of ulcer, and Carlsbad water or salt plays an important rôle in the rest-cure that we have recommended. In speaking of a mineral-water cure, however, in this place, I do not mean the employment of Carlsbad water at home, but at Carlsbad. While I certainly recognize the efficiency of this water, I must reiterate that in the treatment of ulcer the most important factor is the dietetic rest-cure, which may be combined with the administration of Carlsbad water or salt. A dietetic rest-cure cannot, however, be carried out in a watering-place, but only at home, for patients with pronounced ulcer symptoms belong in bed and not in the bath.

In many diseases that are benefited by a course of mineral waters a number of other factors play an important rôle in addition to the curative action of the water—namely, the rest that the patients enjoy, the respite from business care and excitement, life in pure air and in a beautiful region. In ulcer cases all these factors are unimportant, and the chief indications are quiet, rest, and a carefully selected diet. All these advantages can more easily be procured at home than at a watering-place. Later on, after the ulcer is cured, the patients may be sent to Carlsbad in order to combat the hyperacidity that may persist, but an ulcer cure proper should always be carried out at home, and can never be replaced by a course of waters at Carlsbad or any other watering-place.

Steel baths and a sojourn in the mountains or at the seashore will all have to be considered in the after-treatment.

In concluding these therapeutic remarks another method may be briefly mentioned that has been recommended by a number of physicians, namely, washing out the stomach for therapeutic purposes. Schliep was the first to recommend this method of treatment. Following the practice of his day, he employed the stomach-pump. He reports 10 cases of ulcer in which pumping out the stomach was followed by good results. Leube, however, objected to this method on the ground that the sound might easily come in direct contact with the ulcerated portion of the stomach-wall, and in this way lead to perforation or hemorrhage. Although Schliep considered recent ulcers in general as a *noli me tangere*, he still advocated using lavage in very recent cases in which vomiting was severe, and claimed to remove the danger by this method. In a number of cases of this character he obtained the desired result. In chronic ulcers that have either undergone cicatrization or that no longer present pronounced symptoms he considers the pump treatment the most rational method for removing the acid contents of the stomach.

All other authors that express themselves in favor of lavage in these cases recommend it only in old ulcers. Debove is the only one who advises washing out the stomach during the first days, for the purpose chiefly of removing as much of the acid as possible. This physician, in addition, prescribed abundant quantities of alkalis and a non-irritating diet consisting of milk and meat powder, distributed over three meals. Gerhardt states that the best results are obtained from lavage in those old ulcers of the stomach in which there is dilatation of the organ. Bouveret also recommends lavage in old ulcers, particularly in cases that have not had a hemorrhage for a long time. He thinks it is particularly useful if there are retention of food-remnants and hypersecretion.

I do not think that the ulcer *per se* calls for treatment by the sound as long as complications or sequelæ are absent. I consider this method of treatment altogether useless, and believe, moreover, that it may be dangerous under certain circumstances. In uncomplicated ulcer digestion proceeds in an altogether normal manner,—as a matter of fact, more rapidly than normal,—chiefly owing to the condition of hyperchlorhydria that usually exists. Stagnation of food-remnants, therefore, does not occur. Those who think it is necessary, therefore, to remove all acid from the stomach are wrong; all that is needed is to reduce the excess of acid. I do not quite see what good lavage of the stomach could accomplish under these conditions, for we usually remove stomach-contents either because there is decomposition or prolonged stagnation. In simple ulcer neither of these states exists. It would be very unpractical to attempt to combat hyperchlorhydria by aspiration of stomach-contents; all that would be done by this procedure would be to remove the food that is present in the stomach. Hyperchlorhydria can be relieved much more easily and with far less danger to the patient by the methods we have described above.

Ulcer *per se*, therefore, does not call for treatment with the sound. There are, however, a number of cases in which treatment of the stomach with the sound is indicated notwithstanding the existence of an ulcer; these are the cases of stenosis of the pylorus, of motor insufficiency and ectasy, or of hypersecretion, that we so frequently encounter. Here, however, it is not the ulcer that calls for lavage, but the dilatation of the stomach, the hypersecretion, etc. Either condition, if it existed alone, would call for methodic lavage. I refer to the sections on Ectasy, Stenosis of the Pylorus, and Hypersecretion for the exact time when this lavage should be performed, the method of performing it, and other conditions that call for systematic treatment of this character.

I mention the following method, which Take has recommended, merely as a curiosity and to show what peculiar propositions are occasionally made. This author seriously recommended administering calomel by mouth until so severe a degree of stomatitis should develop that the act of chewing and swallowing would become impossible. His idea was to force patients who were suffering from obstinate ulcers of the stomach to stop taking food by mouth and allow rectal feeding exclusively.

Any physician will probably be able to arrange a diet-list with the aid of the tables of nutritive values that I have given in the general part of this work and the general dietary regulations that I have just outlined. I will merely mention a few of suitable dietaries in order to give some general points of view. Numerous variations are permissible, of course, particularly later in the disease. I refer those of my readers who do not wish to prepare their own diet-lists to Biedert's and Langermann's *Dietetics and Cook Book*.

I. Dietary (*for the First Period*).

	Albumin.	Fat.	Carbohydrate.	Calories.
1 quart of milk	34.1	36.5	48.1	676.5
500 c.c. of bouillon	0.5	1.5	. .	14.0
20 gm. of casein-sodium (in milk or bouillon)	16.06	. .	. .	70.0
2 eggs	11.3	10.9	. .	48.1
Total	61.96	48.9	48.1	808.6

II. Dietary (*for the Second Period*).

	Albumin.	Fat.	Carbohydrate.	Calories.
1½ quarts of milk (distributed throughout the day)	51.1	54.5	72.1	1014.5
4 cakes (8 gm.)	3.7	1.4	24.0	126.6
Soup, 15 gm. of sago, 10 gm. of butter, 1 egg	5.83	13.76	12.36	205.0
150 c.c. of meat broth, 1 egg, 10 gm. of casein-sodium	14.29	6.04	. .	83.5
Total	74.92	75.70	108.46	1429.6

III. Dietary (*for the Third Period*).

	Albumin.	Fat.	Carbohydrate.	Calories.
1½ quarts of milk (distributed throughout the day)	51.1	54.5	72.1	1014.5
200 gm. of bouillon, 2 eggs	11.3	10.9	. .	147.0
1 boiled pigeon chopped up[1] (100 gm.)	21.5	1.0	. .	97.0
30 gm. of rice boiled with bouillon	2.0	. .	25.0	110.0
4 cakes (8 gm.)	3.7	1.4	24.0	130.0
Soup from 15 gm. of barley-meal (oats)	1.8	. .	10.5	198.0
10 gm. of butter	. .	8.3	. .	
1 egg	5.6	5.4	. .	
2 Zwieback (20 gm.)	2.4	1.5	13.8	80.0
Total	99.4	83.0	145.4	1776.5

IV. Dietary (*for the Fourth Period*).

	Albumin.	Fat.	Carbohydrate.	Calories.
Tea with 100 gm. of milk	3.4	3.6	4.8	67.0
20 gm. of sugar	. .	. .	20.0	82.0
3 cakes (24 gm.)	2.7	1.1	18.0	98.0
200 gm. of bouillon with 10 gm. of casein-sodium	8.5	. .	. .	159.0
15 gm. of sago	0.1	. .	12.9	
1 egg	5.6	5.4	. .	
Soup from 15 gm. of oat- or barley-meal, 10 gm. of butter, and 1 egg	7.4	13.7	10.5	198.0
150 gm. of beefsteak fried	31.0	7.7	. .	201.0
With 20 gm. of butter	. .	16.6	. .	151.0
100 gm. of mashed potatoes	3.1	0.8	21.0	106.0
Tea with 100 gm. of milk	3.4	3.6	4.8	67.0
20 gm. of sugar	. .	. .	20.0	82.0
3 cakes	2.7	1.1	18.0	98.0
100 gm. of scraped ham	20.2	6.8	. .	146.0
150 gm. of tapioca mush	7.0	5.0	8.0	108.0
20 gm. of toast	1.6	0.2	15.4	72.0
10 gm. of butter	. .	18.3	. .	76.0
250 gm. of milk	8.5	9.1	12.0	169.0
Total	105.2	93.0	165.4	1880.0

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[1] Instead of pigeon, 100 gm. of calves' thymus may be given.

CARCINOMA OF THE STOMACH.

LITERATURE.

For the older literature up to end of the '70's I refer to Leube in *Ziemssen's Handbook of Special Pathology and Therapy.* Of the investigations that have been published since that time, I mention the following:

Van den Velden, "Ueber Vorkommen und Mangel der freien Salzsäure im Magensaft bei Gastrektasie," Deutsch. Arch. f. klin. Med., 1879, vol. xxxiii.

Roger, "Diagnostic et terminaison du cancer du pylore," Thèse de Paris, 1878.

Rilz, "Cancer du pylore," La Presse méd. belg., 1878, No. 7.

Hardy, "Tumeur sarcomateuse de la grande courbure de l'estomac," Gaz. des hôp., 1878, No. 4.

Brissard, "Cancer latent de l'estomac. Pleuresie purulente; pyohémie," Progrès méd., 1878, No. 20.

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See also the monographs and text-books on diseases of the stomach by Boas, Bouveret, etc.

Etiology and Pathogenesis.—No other organ of the body is so frequently the seat of carcinomatous tumors as the stomach. Nearly one-half of all carcinomata occur in the stomach.

According to the statistics of Häberlin, 11,422,—that is, 41.5 per cent.,—of 27,511 cases of carcinoma that terminated fatally were carcinomata of the stomach. These figures correspond essentially with the statement of d'Espine, according to whom 44.3 per cent. of all cancers occur in the stomach. Virchow calculated from the autopsy material in Würzburg, from 1852 to 1855, that the proportion of stomach cancers to cancers of other organs was 34.9 per cent.

No great differences are found in the frequency with which this disease occurs in the two sexes. Fox and Ledoux-Lebard found approximately equal numbers in men and in women. Welsch found a slightly larger number in men. Häberlin's statistics show greater differences, for he found that in men carcinoma is localized in the stomach in nearly one-half of all cases, and in women in only about one-third of the cases. It appears from his summary, however, that after the sixtieth year carcinoma of the stomach is more frequent in women.

Age.—Carcinoma is chiefly a disease of advanced years. According to Häberlin's statistics, about 72 per cent. of all cases occur between the fortieth and the seventieth year. According to the statistics of Hahn, including 166 cases, more than one-half occurred in subjects over sixty years. Eisenhart also states that the maximum of cases occur after the sixtieth year.

Carcinoma of the stomach is a rare disease in youth. The cases of Wilkinson (congenital carcinoma of the stomach), Cullingworth (child of five weeks), and Widerhofer (congenital carcinoma of the stomach) are always mentioned as rarities. Kaulich found a gelatinous carcinoma in a boy of one and one-half years. Lebert examined 314 cases; of these, only 3 were found in subjects under thirty years. According to Schäffer, who described a case of carcinoma of the stomach in a boy of fourteen, the frequency of carcinoma of the stomach in the first three decades is 2 per cent.; according to Reichert, 3 per cent.; according to Bräutigam, 2.5 per cent.

There are marked differences in the geographic distribution of carcinoma of the stomach. In the tropics cancer of the stomach is rare. Griesinger states that he never saw a carcinoma of the stomach in Egypt, and Heizmann states the same for Vera Cruz. According to Häberlin, on the other hand, the disease is very frequent in Switzerland—much more frequent than in Prussia, Austria, or England. It is also said that carcinoma is very common in the Black Forest and in Mecklenburg.

External circumstances and the mode of life do not seem to exercise any appreciable effect on the development of cancer. Carcinoma of the stomach, at all events, is found as frequently in rich people as in persons who live in the greatest poverty. Trade and occupation do not seem to exercise any influence. Antenrieth claims that the disease occurs so frequently in the region of the Bodensee because the inhabitants eat such large quantities of flour, potatoes, and acid articles of diet. As against this view, however, it might be argued that the same diet is eaten in many other regions in which carcinoma of the stomach is not frequent.

I do not think that excessive indulgence in brandy, in cider, or in acid wines (Häberlin) plays any rôle in the genesis of carcinoma of the stomach.

Many authors are inclined to attribute a certain rôle to traumata, for the reason chiefly that in some cases trauma preceded the appearance of the first symptoms of the disease. Brosch has called attention to the fact that carcinomata, if they are not found in the pylorus (their most frequent location), are usually found in the lesser curvature, provided the topography of the stomach is normal. He attributes this to the fact that this portion of the stomach is fixed and anchored by the short mesogastrium, and consequently cannot yield if a blow strikes the stomach; besides, this part of the organ is curved convexly and protrudes into the cavity of the stomach. According to Brosch, a small fold can be found in the lesser curvature in a large percentage of persons who are examined post mortem and whose stomach is found to be normal. This ridge separates the pyloric antrum from the rest of the stomach, and, according to this investigator, forms a typical starting-point for carcinoma of the lesser curvature. In contradistinction to the lesser curvature the greater curvature of the stomach is anchored nowhere, is freely movable, and can consequently recede or yield if a blow or some other injury is inflicted. He argues that carcinoma is hardly ever found in the greater curvature, and that in those cases in which the whole wall of the stomach is infiltrated with carcinoma the greater curvature usually remains free. According to Brosch, those portions of the stomach, therefore, are chiefly involved by primary carcinoma that are particularly exposed to injury. He argues that folds of the stomach-wall or portions of the wall that are pressed against some relatively hard basis by the ingesta are always the ones to become affected.

Other investigators see the primary cause of carcinoma in erosions and scalds of the stomach, and attribute an analogous rôle to these injuries as to mechanical traumata. Here, too, however, according to Brosch, the chemic and thermic lesions are not the direct cause of the development of carcinoma, but the cicatrices and folds that result from these lesions and that create a local predisposition for carcinoma.

The question in regard to the heredity of carcinoma has been much discussed and is not devoid of interest. The statistics that we possess in regard to this matter are naturally incomplete. One of the favorite examples that is frequently quoted in order to prove the heredity of carcinoma is the family of Napoleon, for here carcinoma occurred in several generations. Statistics of this kind, however, to be of value should not only give information in regard to the frequency with which carcinomata occurred in the ancestry of the patient, but they should also give information in regard to the cases of carcinoma that did not transmit the disease to the next generations,—that is, they should tell us how often the children of carcinomatous parents developed carcinoma.

This latter question, however, all our statistics fail to answer even approximately. At all events, the statistics of Häberlin and of Bräutigam do not speak in favor of an hereditary influence in cancer, for the former investigator found only 8 per cent. of cases among 138 that he studied at the Zürich clinic, whose ancestors had suffered from carcinoma of the stomach; the latter, in 120 patients, found a history of fatal stomach trouble of approximately the same duration in one or both parents, only ten times. Schüle obtained similar results, for he found carcinoma of the stomach in the parents or the sisters or brothers in only 6.5 per cent. Häberlin's statistics show that stomach trouble of some kind was present in the parents of his cases in only 17.3 per cent.

In studying the hereditary transmission of carcinoma from parents to children carcinomata of other organs must also be considered. The mother, for instance, may have suffered from a cancer of the breast or the uterus, and the offspring from a cancer of the stomach. There can be no doubt that many cases have been observed in which one of the parents was afflicted with carcinoma and one or the other of the children were subsequently afflicted in the same way. This, however, is not a very frequent occurrence when we consider the relative frequency of carcinoma in general. It is altogether undecided what the particular conditions are that favor the transmission of carcinoma from parent to child. We know as little in regard to this matter as we do in regard to the primary cause of carcinoma. We must refrain from entering into the discussion of the latter question in this place, and refer to the text-books on pathology and pathologic anatomy for the details of this inquiry.

It has frequently been claimed that other diseases of the stomach, in particular chronic inflammatory diseases of the gastric mucosa, increase the predisposition to carcinoma. Statements of this character are frequently encountered, not only in the older works, but also in the recent literature. Clinical observation, however, does not speak in favor of such a connection, for, as a rule, carcinoma develops in individuals who have been entirely well before. If a careful anamnesis is elicited, the statement will usually be made by the patients that they were perfectly well until quite recently, and that suddenly dyspeptic symptoms, loss of strength, emaciation, and similar symptoms developed. A history of a long-lasting chronic catarrh of the stomach is rarely elicited; carcinoma, however, may develop on the basis of an ulcer, or rather of an ulcer scar. Many cases of this kind are quoted in the older literature in which a direct transition of ulcer to carcinoma was observed. Clinically, we are quite frequently able to follow the transition of an ulcer into a carcinoma, or at least to deduce from the anamnesis and the symptom-complex of some previous stomach trouble, together with the objective findings, that an ulcer was primarily present and that now a carcinoma exists, so that it is more than probable that the latter owes its origin to the former. This connection, or, better, this transition, of ulcer symptoms to the symptom-complex of carcinoma can also be determined anatomically. Hauser furnished the proof that atypical proliferation of epithelium can develop from an ulceration; the reports of this investigator have been amply corroborated by subsequent authors.

We do not possess statistics that are sufficiently exhaustive to enable us to determine how frequently carcinoma develops on the basis of an ulcer. Häberlin has calculated from his material in the Zürich clinic that about 7 per cent. of carcinoma cases develop from ulcer; at all events, clinical observation teaches us that this transition is quite frequent.

A certain relation between polyadenoma and cancer has also been discovered. According to Bouveret, only two diseases of the stomach predispose to carcinoma—namely, polyadenoma and ulcer. Adenoma is a glandular hypertrophy in which the general form of the glands is preserved, but in which frequently all transitions up to atypical cancer formation can be seen. These adenomata usually appear in the polypous form, are multiple, and may be present in many different portions of the stomach.

Häberlin found defective teeth in 19 patients,—14 per cent.,—and asks the question whether or not this finding might not help explain the frequency of carcinoma of the stomach in Switzerland. I am inclined, however, to believe that the relative frequency of dental defects together with carcinoma is due to a third common condition—namely, the age of the patients. Carcinoma, as we know, usually affects persons of advanced age, in whom the teeth are, as a rule, defective; consequently the occurrence of defective teeth and carcinoma of the stomach together need not surprise us.

This is essentially all that is known in regard to the causes of carcinoma; it also includes much that has been claimed, but that has no solid foundation in fact. Unfortunately all our knowledge throws very little light on the true origin of carcinoma of the stomach.

It would lead us too far to discuss the different hypotheses that have been formulated as to the origin of cancer. Nowadays even the infectious theory has few adherents. Before this theory was formulated we possessed no positive basis whatever. I might mention that the old idea that carcinoma and tuberculosis never occur together is not correct, for recent experience has taught us that the one disease does not exclude the other, although it is true that the two rarely occur together.

Pathologic Anatomy.—Carcinoma of the stomach, as has been shown chiefly by Waldeyer, starts from the glandular structures of the gastric mucosa. It constitutes an atypical proliferation of epithelium starting from the glandular cells and the epithelia of the efferent ducts of the glands. Atypical proliferation of glands occurs both in diseases of the mucous membrane that lead to atrophy and in the margin of ulcers; it may lead to the formation of polypous excrescences—so-called glandular polypi. Atypical epithelial proliferation in the stomach may also occasionally lead to the formation of infiltrating epithelial tumors —that is, to carcinoma. This epithelial proliferation begins in the mucosa, or rather in the glands of the mucosa; from there it extends to the glandular tissues of the mucous lining and the muscularis mucosæ, the submucosa, the muscularis, and even to the serosa. In the latter cases small isolated nodules develop on the serosa that correspond to the course of the lymph-vessels.

Epithelial proliferation of this character leading to carcinoma may either develop in an apparently normal mucosa or in an atrophic mucous membrane, or finally may start from an ulcer scar.

Very early in the development of the disease neighboring lymph-glands are involved, particularly those that are situated behind the lesser curvature. The carcinomatous new formation may also extend to the mesentery and lead to the deposit of massive carcinomatous nodules. In other cases, again, the carcinomatous neoplasm extends to the venous blood-channels and leads to the formation of carcinomatous thrombi. In still other cases the disease extends to branches of the portal vein, and in this way leads to the development of metastatic foci in the liver, the peritoneum, the pancreas, the kidneys, the lungs, etc.

Carcinoma of the stomach is most frequently seen in the pyloric region. Lebert has arranged the following table to show the frequency with which carcinoma is localized in different portions of the stomach:

Pylorus	51 per cent.	Posterior wall	4 per cent.
Lesser curvature	16 per cent.	Anterior and posterior wall	4 per cent.
Cardia	9 per cent.	Greater curvature	4 per cent.
Anterior wall	3 per cent.	Diffuse infiltration	6 per cent.

It appears, therefore, that the pylorus is the chief point of predilection for cancer; second in importance is the lesser curvature, and third, the cardia. These portions of the stomach are, on the one hand, those that are chiefly exposed to mechanical injuries; on the other hand, those in which protrusions of the mucous membrane, folds, ridges, etc., occur. In addition it must be remembered that these parts of the stomach are firmly anchored and fixed so that their motility is slight. Israel, however, has shown by a number of postmortem examinations that many carcinomata that on autopsy are found to be carcinomata of the pylorus do not necessarily originate from the ostium, but are simply arrested in their further growth as soon as they reach this portion of the stomach. It appears that the lesser curvature of the stomach is a frequent starting-point of carcinoma, probably because its motility is very much impaired, owing to the shortness of its mesogastrium.

Carcinoma of the stomach is usually a primary disease. The stomach is rarely the seat of secondary metastatic carcinomata or of carcinoma that extends to it by continuity or contiguity from some neighboring organ. Of the different organs that are in close proximity to the stomach, the pancreas is the most frequent seat of the primary carcinoma.

Carcinoma of the stomach usually appears as a tumor of varying size and protrudes into the lumen of the organ. If it is situated in the pyloric region, it usually narrows this part of the stomach. As soon as the neoplasm grows to a certain size necrotic disintegration of its central portion, as a rule, occurs, and a carcinomatous ulcer is formed. This form of ulcer is distinguished from other forms chiefly by its ridge-like margin. The base of such an ulcer is ragged, usually dark in color, and occasionally thrombosed vessels are seen there. Here and there cicatrization occurs, but in almost all cases there is a tendency to further extension. The base of the ulcer is either formed by tissue that shows carcinomatous infiltration or by submucosa or muscularis that is thickened owing to fibrous hyperplasia, or, in rare instances, by the serosa itself.

The time at which this disintegration of the carcinomatous growth occurs varies. Sometimes small tumors are found in a state of disintegration, or the neoplasm attains considerable dimensions and extends far down into the stomach and over a large area of its wall before disintegration and ulcer-formation occur. Occasionally the tumor disintegrates completely, so that the surface of the ulcer appears perfectly smooth. If in a case of this kind carcinomatous nodules are not seen in the submucosa, the muscularis, and the serosa, and if these tissues are merely hardened by connective-tissue hyperplasia, the impression may be created that the lesion is a benign fibrous induration. Ziegler has called attention to the fact that cases of carcinoma of the stomach may occur in which even microscopic examination fails to reveal foci of cancer-cells anywhere, so that the only way in which we can determine that the lesion is cancerous is by studying the metastases that may be present.

Different forms of cancer occur in the stomach. It is important both from an anatomic-pathologic point of view and from a clinical point of view to separate the different forms of cancer that can occur in this region, for the clinical course may vary according to the nature of the carcinoma. The following main varieties can be distinguished:

(a) **Carcinoma Medullare** (**Medullary Carcinoma; "Markschwamm"**).—This growth consists of spongy nodules that are usually situated in the pyloric portion of the stomach. Histologically, medullary carcinomata are characterized by the abundance of the cells, the large number of cancer-cell nests, and the slight development of stroma. Partial disintegration with the formation of ulcer occurs frequently and quite early in the development of the growth. Metastases in neighboring organs—the liver, the peritoneum, etc.—are not rare.

(b) **Carcinoma Fibrosum** (**Scirrhus; "Faserkrebs"**).—This growth is characterized by a large quantity of connective tissue containing few blood-vessels. Owing to the presence of this connective tissue the tumor feels hard and tough. As a rule, it develops slowly. It occurs either in the form of a diffuse thickening and hardening of the stomach-wall in the pyloric portion of the organ or in the form of isolated areas of infiltration that extend to the serosa. In this form of cancer small coarse carcinoma nodules are quite frequently found on the serous lining of the organ. Owing to the tendency of the connective tissue contained in this tumor to contract, stenosis of the pylorus occurs quite frequently if the growth is situated in this portion of the organ; if the lesion is diffuse and scirrhous the whole organ may become diminished in size and contracted, so that the central cavity becomes much smaller than normal (cancer atrophicans). The macroscopic picture of this condition frequently resembles the cirrhosis of the stomach that we have described above; only microscopic examination enables us to differentiate between the two forms. Occasionally ulcers may form, but this does not occur so frequently in this form of cancer as in the one described above.

(c) **Carcinoma Adenomatosum** (**Adenocarcinoma; Epithelioma**).—This kind of cancer forms soft tumors that disintegrate readily and lead to the formation of ulcers. Hemorrhages occur quite frequently, also metastases. Histologically, this form is characterized by tubular, gland-like structures; gradually these tubular structures develop into large cell-nests; at the margin of these nests cylindric epithelium is seen, whereas the interior is filled out by polymorphous cancer-cells. The stroma is only slightly developed and frequently shows small-celled infiltration.

(d) **Carcinoma Gelatinosum or Colloides** (**Mucoid or Gelatinous Carcinoma**).—This form is rare. The cancer appears either in the form of nodular tumors or in the form of a diffuse infiltration of the stomach-wall. The neoplastic tissue consists almost exclusively of gelatinous material, or at least contains foci that are translucent, like gelatin. These gelatinous masses originate in part from the cancer-cells themselves, in part from the connective-tissue stroma (Ziegler).

(e) **Carcinomata Consisting of Squamous Epithelium.**—This form is found only in the cardiac region as the continuation of a carcinoma of the esophagus.

Various transition forms from one variety of cancer to the other are frequently encountered.

Those portions of the gastric mucosa that are not directly involved by the cancer in pronounced cases usually show more or less atrophy. The picture, of course, will vary in different cases according to the stage and the extent of the cancer.

Hammerschlag[1] has published some very careful investigations into the abnormalities of the gastric mucosa in carcinoma of the stomach. These investigations are important, particularly because they were performed on pieces of gastric mucosa that were removed from cases of gastro-enterostomy or resection of the pylorus. The tissues, therefore, were always fresh. In all these cases the chemism of the stomach was carefully investigated in addition.

As was to be expected, the results of these examinations varied. In cancer of the stomach, in which the hydrochloric acid secretion of the organ was completely intact, no structural changes of the gastric mucosa were observed. In cases, on the other hand, in which the peptic power of the stomach was very much reduced, in which free hydrochloric acid was absent, in which there was formation of lactic acid, a different picture was presented. Hammerschlag could corroborate the statement of Schmidt in regard to these cases—namely, that the superficial epithelium of the gastric mucosa, as a rule, remained intact. The most important change was a disappearance, within circumscribed areas, of the specific glandular elements of the stomach. Within these areas the glandular elements were replaced by cylindric epithelium. Specific rennet glands were, as a rule, completely absent; in their place were seen tubules that were lined with cylindric epithelium. These tubules ran either vertically or diagonally, and divided and subdivided, thus forming numerous arborescences and diverticula. On superficial examination of transverse sections through these tubules the latter might have been mistaken for glands; on more careful examination, however, it could usually be determined without difficulty that these structures were not rennet glands, for the epithelium that lined the tubules was cylindric epithelium. This could be seen very distinctly in those specimens in which the tubules were lined with goblet cells and epithelium that had undergone mucoid degeneration. The metamorphosis was seen particularly in the region of the pylorus. The last-named change has frequently been described as mucoid degeneration of glandular epithelium, but Schmidt has shown that the epithelia that are degenerated are not real glandular epithelia, but are altogether different from the epithelia of the rennet glands. He showed that they are identical with the surface epithelia of the gastric mucosa.

Schmidt is inclined to believe that these structures are true mucous glands. Hammerschlag is inclined to consider them prolonged and proliferating gastric follicles. According to Hammerschlag, the specific rennet glands perish and desquamate; the cylindric epithelium of the follicles, however, proliferates so that it replaces the rennet cells. The new epithelium, of course, is not concerned in secretion.

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[1] *Arch. f. Verdauungskrankh.*, vol. ii.

In many specimens nothing can be seen of the glandular layer of the mucosa. The mucous lining of the stomach consists of neoplastic cicatricial connective tissue and of a swollen meshwork that incloses round-cells and a number of isolated fragments of glands. In addition, of course, the follicles of the stomach are intact. In other cases again the rennet glands disappear in certain circumscribed areas, so that here and there the glandular layer remains intact, but is usually very much narrower than normal. The tubules of the glands within such areas no longer pursue a parallel course, but run diagonally, are bent, and frequently dilated to form cysts. Sometimes they are cystic; usually they are separated from one another by an area of small-celled infiltration. The structure of the glands usually presents a normal appearance, even in those places in which the tubules are changed. If glands are present at all, the peptic and parietal cells can be stained, and it will be seen that the number of parietal cells is very much reduced; in fact, that these cells may be completely absent in certain places (Hammerschlag). The interspaces between the rows of glands are usually filled out by an interstitial small-celled infiltration or by neoplastic connective tissue. Sometimes the rennet glands will be destroyed over large areas, and at the same time the interstitial changes in the mucosa be very slight; in other cases, again, the latter are very pronounced.

Hammerschlag also succeeded repeatedly in finding eosinophile cells among the round-cells lying in the interstices. In some instances these eosinophile cells were very numerous. Schmidt and Cohnheim reported the discovery of eosinophile cells in atrophy of the gastric mucosa. The significance and the origin of these eosinophile cells are not yet established.

We see, therefore, that in carcinoma of the stomach there is a focalized destruction of rennet glands, involving particularly the parietal cells. The epithelia that are lost in this way are replaced by the proliferating epithelium of the follicles; in addition there are small-celled infiltration and neoplastic formation of connective tissue in the gastric mucosa.

This, then, is a secondary atrophic gastritis. As a rule, the process is most pronounced in the region of the pylorus. We can readily understand how this condition can cause a loss of peptic power. There is a great diversity of opinion in regard to the connection between this atrophy of the gastric mucosa and carcinoma. The majority of authors are inclined to consider the atrophy a sequel of carcinoma. Mathieu, and later Hayem, however, have expressed the opinion that the reverse is the case—that carcinoma develops secondarily on the basis of "*gastrite apeptique.*"

This view, however, is not corroborated by the clinical course of the disease. Ordinary atrophy is a process that develops slowly. In carcinoma we usually see apepsia develop within a short time, and, as a rule, it is not preceded by gastritis that might lead to atrophy.

Another argument that speaks in favor of the view that changes in the mucosa are secondary to carcinoma is the fact that the secretory function of the stomach, provided it is not completely lost, does not disappear after resection of a carcinoma; that, in fact, in some instances free hydrochloric acid reappears in the gastric juice, whereas in gastroenterostomy the secretory function of the stomach progressively decreases and finally disappears altogether (Mintz). Fenwick, in addition, has demonstrated that atrophy of the gastric mucosa may occur in carcinoma of other organs—above all, in the breast and the uterus. In this case, of course, atrophy must be secondary.

Other peculiarities that are observed in the stomach in carcinoma are the following: If the carcinoma is situated at the pylorus; if it leads to stenosis of the lumen of the pylorus, the stomach will, as a rule, become more or less dilated. The muscularis, particularly in the region of the pylorus, is, as a rule, more or less hypertrophic, provided, of course, it is not infiltrated with carcinoma. Very frequently the stomach, in addition to being dilated, is also found low down in the abdomen or in a vertical position. If adhesions form between the pylorus and the neighboring organs, this downward movement is, of course, prevented. If the carcinoma is situated near the cardia and leads to stenosis of this orifice, the stomach will frequently be found abnormally small and the esophagus at the same time dilated above the stenosis. [Boas has noted the presence of food stagnation when the carcinoma is located in the fundus; the cause of this is not clear, but unquestionably it sometimes occurs. As a rule, when there is absence of stagnation, the patient suffers comparatively little discomfort until near the end of his life.—Ed.] Other forms of adhesions, of course, may lead to different forms of dislocation of the stomach, or may produce hour-glass contraction of the organ. I need hardly explain this in detail. Insufficiency of the pylorus may also develop if the pyloric muscle is destroyed by cancerous infiltration.

Carcinoma of the stomach rarely leads to free perforation into the abdominal cavity. This is due to the fact that carcinomata, as a rule, develop very slowly, so that sufficient time is given for the formation of adhesions with neighboring organs; if, nevertheless, free perforation occurs, the well-known symptoms of severe diffuse septic peritonitis develop. Perforation may also occur into some cavity that has been formed by adhesions; or, again, an abnormal communication may be formed between the stomach and some other viscus—the intestine, the gall-bladder, etc. Carcinomata may also perforate through the diaphragm or through the abdominal walls; in the latter instance an external gastric fistula will develop. Eighteen cases of abdominal fistula following carcinoma are on record. The perforation through the skin was frequently preceded by cutaneous emphysema.

In contradistinction to ulcer, cancer of the stomach rarely leads to the formation of subphrenic abscess or the development of pyopneumothorax. Perforation into the pleural cavity, the heart, or the pericardium is very rare. In a few instances the carcinoma extended to the spinal column and even involved the meninges of the spinal cord.

Other sequelæ that are occasionally encountered are erosion of large blood-vessels, thrombosis, and emboli.

Symptoms.—The clinical picture of carcinoma of the stomach presents many variations according to the stage, the extent, and the localization of the tumor. Carcinoma of the stomach is most frequently found in the region of the pylorus. Carcinoma is found in the latter location more than twice as often as in all other locations together. Carcinoma of the pylorus, therefore, is the rule; of other regions of the stomach, the exception. For this reason I will describe the picture of this frequent form of carcinoma of the pylorus first, and later on discuss the deviations from this syndrome that are seen if the cancer is located in other portions of the organ.

The Disease-picture in General.—The symptom-complex varies according to the stage in which we examine the patient and according to certain special conditions that may vary in individual cases. Occasionally we merely find symptoms that are found in any form of simple gastritis; or, again, we encounter so pronounced a symptom-complex that the disease can be recognized at once even by an inexperienced observer.

Let us begin by discussing the latter picture first—namely, that of a carcinoma of the pylorus in an advanced stage.

We will assume that a man of fifty or sixty comes to the clinic and gives the following history: The patient was always perfectly well and enjoyed a good appetite. His present trouble began, according to his statement, about six months ago. There was gradual loss of appetite, and after eating he complained of a slight feeling of distress, which later on grew more severe. At the same time there was a feeling of pressure and fulness in the region of the stomach, frequently combined with belching. Notwithstanding the most careful diet his condition did not improve; on the contrary, his symptoms seemed gradually to grow worse. Gradually, too, the patient tells us, there have been a loss of strength, a loss of energy, a distaste for work, and general depression. The bowels have been constipated. Within the last two months all these symptoms have become aggravated. There is at present complete loss of appetite, and particularly a distaste for any form of meat. The patient, too, has become more and more emaciated of late. There has been an occasional attack of vomiting, and the vomit consists partly of undigested acid masses of food and partly of a coffee-ground-like material.

On examination we find that the patient is very much emaciated, that his complexion is sallow and cachectic, that the skin is flaccid and wilted, and the muscles thin and atrophic. On examining the abdomen we find the region of the stomach distended and can distinctly palpate the boundaries of the dilated stomach through the thin relaxed abdominal wall. The lower boundary of the stomach is found two fingers below the umbilicus. Inspection and other methods of examination, particularly inflation, corroborate this. From time to time peristaltic waves run over the stomach from left to right. A slight succussion sound can be elicited over the whole extent of the dilated organ by mild tapping. The region of the stomach is somewhat sensitive to pressure, but it is impossible to discover any strictly circumscribed painful pressure-point. To the right, however, underneath the costal arch, in the region of the pylorus, we feel a small, hard, nodular tumor that is somewhat sensitive to pressure. We aspirate the stomach-contents and evacuate an abundant quantity of acid material, consisting of partially digested coarse particles of food. An analysis of the filtrate reveals the absence of all free hydrochloric acid, but demonstrates the presence of lactic acid. After thoroughly washing out the stomach we administer a test-meal; the patient will eat with reluctance and take only a part of the test-meal. At the expiration of six hours we aspirate the stomach-contents again, and again find a relatively abundant quantity of residue containing numerous coarse morsels of meat. An analysis reveals the absence of free hydrochloric acid and the presence of lactic acid; the same result is obtained after a test-breakfast.

If aspiration of the stomach-contents is repeated several times in a case of this character, and if the same result is obtained in every instance, no one will doubt for a moment that a patient is suffering from carcinoma of the pylorus with secondary ectasy of the stomach.

If we include early stages of the disease and those cases in which the carcinoma is localized in other portions of the stomach, the majority of cases will not present so pronounced a symptom-complex, so that it is impossible to render a diagnosis unless the patient is observed for a long time; in many of these cases, however, a diagnosis can be made if we weigh carefully all the evidence. The following case illustrates this:

About three years ago one of my colleagues consulted me. He stated that he had been suffering from dyspeptic disturbances for about half a year. He complained of a feeling of pressure and fulness in the region of the stomach after eating, of loss of appetite, and of frequent belching. Vomiting, he said, had never occurred. The patient was forty-two years old. An objective examination revealed that he was well nourished, that there was an abundant adipose layer, and that there was no marasmus nor cachexia. The tongue was slightly coated; the region of the stomach did not protrude, but was slightly sensitive to pressure; nowhere, however, could any circumscribed painful pressure-point or any abnormal resistance be discovered. The organ was not enlarged. Analysis of the stomach-contents, however, revealed that a relatively abundant quantity of residue containing coarse morsels of meat and bread could be aspirated after a test-meal. Free hydrochloric acid could not be discovered in the filtrate; lactic acid, however, was present. I might add that the patient was very nervous, and that the physician who was treating him had made a diagnosis of nervous dyspepsia, without, however, having analyzed the stomach-contents.

The only objective symptoms, therefore, that were discovered, were an unexpected reduction of peptic power, the presence of lactic acid, and reduced motility, without pronounced ectasy. Although this patient, therefore, was well nourished and although his general strength was good, the suspicion of carcinoma was justified, particularly as the disease was of such short duration.

I did not have an opportunity to examine this case for several weeks. When I saw him for the second time the general nutrition was better and the general strength improved. The patient was very hopeful and looked forward to an early recovery. The examination of the stomach-contents gave the same result this time as before.

The further course of the disease, briefly, was as follows: Five months later I succeeded for the first time in discovering a small nodule in the epigastrium; from that time on this tumor increased rapidly in size. The physician who had charge of the patient in another city reported to me that the tumor continued to increase very rapidly. The patient began to emaciate and to lose strength. An attempt was made to remove the tumor, but it was found that numerous adhesions and metastases were present, so that the operation was not undertaken. The patient did not die until a year later.

This case illustrates how indefinite the symptom-complex of carcinoma of the stomach may be in the earlier stages of the disease. It seems to me particularly interesting to remember that free hydrochloric acid was absent and lactic acid present at a time when there was no emaciation, no cachexia, and no palpable tumor. In this case death did not result for a year and a half after this typical anomaly was discovered.

We are justified in assuming from the general course of this case that the carcinoma at this time was very small. At all events, we learn that the peptic powers of the stomach may be much reduced in very early stages of carcinoma—that is, at a time when the tumor is small and when the general nutrition of the patient is not much reduced. This case is not exceptional by any means. It is true that cases of this character frequently remain unrecognized, for the reason, chiefly, that the mildness of the symptoms in the initial stages of the disease leads many physicians to think that an examination of the stomach-contents is not necessary. Patients who are suffering from carcinoma in these early stages are rarely seen in hospitals—more frequently in private practice.

I had occasion to examine a second case of carcinoma at the same time. This patient also stated that he had been perfectly healthy up to a few months ago. Following an attack of influenza, dyspeptic symptoms, loss of appetite, a slight feeling of pressure in the region of the stomach, and frequent vomiting developed. As these symptoms seemed to persist, the patient consulted me. The man was forty-five years old. On objective examination it was seen that he was very strong and well nourished; ectasy and tumor were absent; there was no circumscribed area of pain, and only slight sensitiveness to pressure in the region of the stomach. An examination of the stomach-contents after a test-meal showed that there was a relatively small amount of residue, but that it consisted of coarse morsels of food, particularly meat, that were almost undigested. Here, too, every trace of free hydrochloric acid was absent. The lactic acid reaction was not distinct. In this case, therefore, the motor power was still good, but the peptic power was very much reduced. The stomach-contents were repeatedly analyzed at intervals of about a week. The analysis always gave the same result.

I felt justified in excluding the severe or the atrophic form of gastritis in this case, because the whole disease was of such short duration. Nothing, moreover, indicated nervous dyspepsia nor amyloid degeneration of the gastric mucosa. No tumor could be felt for a whole year. At the expiration of this time a small tumor was discovered in the lesser curvature. Neither vomiting nor hematemesis occurred throughout the whole course of the disease. The patient died two years after the onset of the initial symptoms. On autopsy the diagnosis—carcinoma of the lesser curvature—was corroborated.

These few examples show how different the disease-picture may be, and what a marked influence the stage of the cancer and the localization of the tumor exercises on the general symptom-complex. We must remember that the course of carcinoma is always progressive; that the disease begins with mild and insignificant symptoms, and ultimately leads to severe symptoms that gradually impair the general health of the patient.

The course of carcinoma of the stomach is most typical if the cancer, as is most frequently the case, is situated in the pyloric region. Here, too, however, the symptoms are not at all characteristic in the beginning, and resemble those of simple gastritis throughout. Sooner or later, however, more or less severe degrees of motor insufficiency and ectasy develop; even if no tumor can be found in the beginning, it can usually be discovered some time during the course of the disease; later coffee-ground material is vomited, the patient gradually goes into a decline, emaciation becomes extreme, the appearance of the patient grows cachectic, and death results from advanced marasmus and loss of strength. In some instances intercurrent diseases or complications accelerate the fatal issue. This is the ordinary course of this disease; many cases, however, are encountered in which the course is different.

The duration of the disease averages from one to two years. We must never forget, however, that the onset is rarely marked, so that we cannot, as a rule, determine the time of onset with certainty. It is probable, therefore, that the duration of the disease is really longer than is generally assumed.

Individual Symptoms.—*Onset.*—The onset of carcinoma of the stomach, no matter in what portion of the organ it may be located, is, almost without exception, gradual. The first symptoms usually consist in mild dyspeptic disturbances. The disease occurs most frequently in individuals of advanced years—between the years of fifty and sixty. Generally the patients state that they were perfectly healthy until a short time ago, and never suffered from any dyspeptic disturbances. The first thing that the patients usually notice is a loss of appetite; they complain of a feeling of fulness and pressure in the stomach region, even after eating small quantities of food. Belching is also frequently complained of. The symptoms are essentially those of a mild gastritis. Occasionally, however, the onset of the disease is sudden, and a number of cases of this kind are recorded in the literature (Hammerschlag and others). The patients themselves frequently attribute the onset of the disease to an acute attack of indigestion, to influenza, or to some other acute disease. The case of carcinoma that we mentioned above stated that his disease had begun with influenza, and his physician himself considered the disease a sequel to influenza. As a result the latter did not realize how serious the condition was even though there was constant absence of free hydrochloric acid. If we investigate cases of this kind more thoroughly, we will usually discover that the patients suffered from mild dyspeptic disturbances before the onset of the acute disease. Generally these preliminary dyspeptic disturbances are so mild that the patients hardly notice them. As soon, however, as the acute disease is contracted, they seem to exacerbate and to persist with greater severity after the acute disease is over.

Those cases of cancer that develop from an ulcer usually form an exception to this rule. Here it is hardly possible even to estimate when the disease began. The anamnesis, as a rule, indicates that cardialgia and other symptoms of ulcer existed for a long time—sometimes for years. Occasionally it is stated that these ulcer symptoms intermitted. If cancer is developing, however, the patients will state that of late dyspeptic disturbances of a less typical character appeared. Sometimes these vague symptoms appear after a period of apparent health in which all dyspeptic symptoms were absent.

If the carcinoma is situated in the cardia the initial symptoms do not vary in any respect from those we have described above. Here, too, the patients complain of a feeling of pressure in the region of the stomach; they frequently describe a peculiar sensation—namely, as though a foreign body were lodged in the region of the stomach. Vomiting and regurgitation of food do not occur until the lumen of the cardia is narrowed and diverticula have formed above it. True pain is more frequently felt in carcinoma of the cardia than in carcinoma of the pylorus or of the body of the stomach. In carcinoma of the cardia this pain is felt in the region of the lower end of the sternum; it appears not only after eating, but altogether independently of swallowing. There are, however, many cases of carcinoma of the cardia that run an altogether latent course, until finally symptoms of stenosis appear. We must regard it as a general rule that carcinoma of the stomach, wherever it is located, develops gradually and slowly.

Appetite.—In the beginning there are only mild dyspeptic disturbances, just as in simple gastritis; gradually these symptoms increase in severity and obstinately resist all treatment. As a result there is a progressive loss of appetite; the patients experience a disagreeable feeling of pressure and fulness in the region of the stomach even after eating very small meals. In exceptional cases the appetite remains good for some time. In rare instances we are able to improve the appetite during the course of the disease, but we will find, as a rule, that it is lost again sooner or later. Cases of carcinoma of the stomach with a good appetite are certainly rare. Hanot claims to have observed that many of his patients enjoyed a good appetite during the greater part of the disease; unfortunately Hanot claims that not only was the appetite good, but that digestion was normal, so that we cannot consider his statements convincing, and must assume that his method of examining the peptic power of the stomach in these cases was deficient.

Dujardin-Beaumetz reports the case of a cancer patient who was able to eat the most indigestible food without distress, and Huchard, in 1880, together with Peter, observed a patient who presented all the symptoms of a carcinoma of the stomach excepting a palpable tumor, and whose appetite remained good throughout the whole course of the disease, so that the diagnosis for this reason remained doubtful for some time. Arnozan tells us of a patient with cancer of the stomach whose appetite remained good until death, notwithstanding the presence of a large tumor and of pronounced cachexia. Unfortunately, these authors failed to make careful statements in regard to the secretory and motor powers of the stomach.

To judge from my own experience, it appears that in the early stages of the disease, particularly in those cases where the motor power of the stomach remains undisturbed, the appetite may remain good. The same applies to those cases in which carcinoma develops on the basis of an ulcer or an ulcer scar.

[In a recent article upon the study of 141 cases of gastric carcinoma, Boas[1] noticed that in 33 there was a fair or an increased appetite, and that sometimes it was excessive. An interesting fact to note is that in 33 per cent. of all cases when one may expect to find a depreciation, he, on the contrary, found an increase in the desire for food. He thinks that a common cause of poor appetite is lack of care of the mouth and tongue. When these are carefully attended to, the appetite is likely to be fairly good.—Ed.]

Bulimia is rarely observed. Hanot reports a case of carcinoma of the stomach in which the first symptom of the disease was bulimia, which lasted for two months. During this time, we are told, the patient ate enormous quantities of food. Cases of this kind, however, are very rare. As a rule, there is severe anorexia, particularly aversion to meat; in general, all attempts to stimulate the appetite by stomachics fail. Patients who are in the habit of smoking a good deal, as a rule, develop an aversion for tobacco.

Many patients also complain of belching and vomiting. If in the course of a stenosis of the pylorus secondary ectasy develops, all the symptoms we have described become more severe.

The Pain.—Carcinoma of the stomach is usually accompanied by pain, and very few cases are observed in which pain is not present at some time during the disease. Generally speaking, however, the pain plays a less important rôle in carcinoma than in ulcer; the pain hardly ever assumes the character of the cardialgic attacks that are seen in ulcer patients. In general, the patients merely complain of a feeling of discomfort or of a painful dull feeling in the region of the stomach; the pain, moreover, is rarely limited to any small circumscribed area. It will frequently be found that it is most intense in the region where the cancer is situated.

As in all other affections of the stomach that are accompanied by pain, we must differentiate between the pain that occurs spontaneously and the pain that is caused only by pressure; we must further determine whether it occurs at a certain time during the period of digestion or at irregular intervals. Many patients complain of a constant feeling of pressure and discomfort in the region of the stomach, and state that the distress increases as soon as they eat anything. Violent pain is absent in many cases; more frequently we find that the region of the stomach is sensitive to pressure over a large area; in other instances we find that only that portion of the abdomen that corresponds to the location of the tumor is sensitive to pressure. It is possible in many cases to predict the exact portion of the epigastrium in which pain will be felt on pressure if we know the portion of the stomach that is involved. This prediction usually surprises the patient very much.

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[1] Boas, *Arch. f. Verdauungskrankh.*, vol. vii., Nos. 4, 5.

In those cases in which pain occurs only after eating, or in which it is exacerbated after the ingestion of food, it rarely occurs immediately after the food is swallowed, but, as a rule, later. The pain usually appears later than the cardialgic attacks of ulcer. If the carcinoma is situated in the cardia, pain is frequently felt at once, particularly when solid food is swallowed. Sometimes the pain from carcinoma of the cardia is continuous and independent of swallowing.

The pain occasionally radiates in different directions, particularly into the back. If the carcinoma is situated in the cardia, the pain quite frequently is transmitted upward along the sternum. The character of the pain varies: there may be either a vague feeling of distress in the region of the stomach or there may be a dull, burning sensation. The pain is not characteristic in any way. It is usually less severe in carcinoma than in ulcer. If it is increased by eating, it rarely becomes as severe as in ulcer. If very mild pressure over the carcinoma elicits much pain, we must always think of some complication, particularly of circumscribed peritonitis.

Many patients complain of a frequently recurring feeling of unrest—of spasmodic movements of the stomach. This is observed particularly in cases in which the carcinoma is situated in the pylorus and has led to a stenosis of this portion of the stomach. In very thin subjects these peristaltic movements of the stomach can quite frequently be seen through the thin abdominal walls. They occasionally persist for some time, and do not stop until the contents of the stomach is evacuated—either upward by vomiting or by aspiration, or downward into the intestine.

Vomiting is very frequently observed in carcinoma of the stomach. As a rule, this symptom appears at a late period of the disease. Vomiting is seen particularly in those cases in which the carcinoma is situated in the region of the pylorus; in the latter instance stenosis of the lumen of the pylorus occurs, so that the evacuation of the stomach-contents into the intestine is rendered difficult and ectasy develops. Vomiting in these cases is frequently very obstinate; it may recur daily or only once every few days; in general, abundant masses of coarse, undigested, acid material are raised, frequently containing remnants of food that was eaten several days before. If the carcinoma is situated in the cardia and has caused stenosis of this orifice, the character of the vomiting is slightly different; here we do not see real vomiting, but merely a regurgitation of food. The material, as a rule, contains abundant quantities of mucus that come from the esophagus. In contradistinction to the vomiting in carcinoma of the pylorus, the vomiting in carcinoma of the cardia occurs with greater regularity, and, as a rule, very soon after eating.

There are cases of carcinoma in which vomiting never occurs or occurs only for a short time during the course of the disease. I have a patient, for instance, at the present time who has never thus far vomited, although the diagnosis of carcinoma has been established for more than seven months. In this case there is no evidence of ectasy, and the carcinoma is situated at the greater curvature of the stomach and not at the cardia or the pylorus. The location of the carcinoma determines whether or not vomiting will occur; if the tumor is situated in a portion of the stomach where it does not interfere with the passage of the ingesta,—for instance, at the lesser or the greater curvature or at the posterior wall,—it rarely causes vomiting. The size and the development of the neoplasm may also exercise an influence in this direction. We occasionally see, for instance in carcinoma of the pylorus, that vomiting is very obstinate in the beginning, gradually recurs with less frequency, and finally stops altogether. This is probably due to disintegration and softening of the carcinoma, which leads to a widening of the lumen of the pylorus. We see analogous conditions in carcinoma of the cardia and the esophagus. Here it is often impossible to pass even a very small sound for some time; suddenly the passage will become free, so that relatively large sounds can pass the stenosed portion of the stomach.

The most important cause of vomiting in carcinoma of the stomach is stenosis of the orifices of the organ, particularly of the pylorus. In the latter condition there is stagnation of ingesta, and this is a prolific cause of vomiting. This view is strengthened by the fact that methodic daily lavage, which removes all remnants of food, frequently stops the vomiting in these cases.

Other factors may be concerned in the production of vomiting—for instance, a complicating gastritis.

The time at which vomiting occurs varies. In carcinoma of the pylorus it occurs with greatest frequency in the late hours of the afternoon or in the evening; it may, however, occur at any other time of the day. In many instances vomiting occurs only every few days, whenever the stomach becomes distended to a certain degree. Vomiting usually occurs easily, without much retching.

The quantity of vomit raised is frequently very large, and may amount to half a liter, one liter, or even more. The appearance of the vomit naturally varies according to the food; in general, however, the vomit in carcinoma, as we have already said, is characterized by the presence of coarse or undigested particles of food. Occasionally remnants of food are found that were eaten several days before. Mucus is an occasional admixture; bile is rarely present in the vomit. Blood is more frequently seen, though usually in small quantities; in the later stages of the disease blood is more common.

A microscopic examination of the vomit reveals approximately the same conditions as an examination of the aspirated stomach-contents; we refer, therefore, to the section on Microscopic Examination of the Stomach-contents. We also refer to the section on the Chemical Analysis of the Vomit for the details of this examination.

Vomiting of Blood.—We have already mentioned that blood is quite frequently present in the vomit. As a rule, however, the bleeding in carcinoma, in contradistinction to ulcer, is a slight parenchymatous hemorrhage; this is natural when we consider the tendency of carcinomata to disintegrate. In exceptional cases a large artery may become eroded and a violent, even fatal, hemorrhage result.

Brinton has calculated that hemorrhage occurs in 42 per cent. of all cases; I think, however, that this figure is too low rather than too high. Mild degrees of hemorrhage easily avoid detection because they do not produce vomiting. In performing lavage of the stomach in cases of carcinoma, black bloody material is frequently found in cases in which all symptoms of hemorrhage are absent. If the stomach-contents had not been removed by lavage, this hemorrhage would not have been discovered. As the loss of blood is usually slight, and as the bleeding occurs slowly, the blood remains in the stomach for a considerable time and consequently becomes changed; this explains the chocolate or coffee-ground appearance of the vomit in so many cases of carcinoma. If we are not certain that the dark color of the vomit is due to blood, the stomach-contents should be examined according to the methods we have described in the general part of this work for detecting small quantities of blood.

The bowels, as a rule, particularly in the beginning, are inclined to be sluggish; in the later stages of the disease diarrhea is occasionally observed, or diarrhea may alternate with constipation. Tripier claims to have observed diarrhea in more than one-half of the cases, frequently during the last months of life. According to this author, the consistency of the stools depends largely on the amount of food eaten; if considerable quantities of food are eaten, the gastro-intestinal tract becomes irritated as a result of the insufficiency of the gastric functions, and in this way diarrhea is very apt to occur; if, on the other hand, the patients eat little or vomit frequently, diarrhea is not so apt to occur.

The objective signs of carcinoma of the stomach are much more important than all the symptoms we have described, for the latter are mainly determined by the anamnesis.

If we follow the ordinary course of an objective examination, we will first study the state of the general nutrition of our patient. As a rule, we are told that cancer cases are emaciated, have a flaccid, sallow skin, and flaccid, atrophic muscles; the panniculus adiposus is absent; the complexion ashy and cachectic; the patients appear dried out. All this is undoubtedly true in the advanced stages of the disease. Occasionally, however, we encounter patients who are very pale, although their general nutrition is still relatively good; this condition is principally due to repeated gastric hemorrhages. Sometimes this appearance is seen in patients in earlier stages of the disease when they should look different. It is important to recognize the disease precisely in these cases; for, if we diagnose cancer only when the patients look cachectic, are extremely emaciated and very weak, we shall hardly ever be able to help them. It is not surprising to find cancer patients in the last stages very much emaciated and weak, for this is the natural result of the almost complete loss of appetite, of insufficient nourishment, of the reduced peptic powers of the stomach, and the disturbances of the motor powers of the organ. In carcinoma still another factor must be considered. In the majority of stomach diseases the loss of strength, as a rule, runs parallel to the decreased ingestion of food. The investigations of F. Müller, Klemperer, and others have shown that in carcinoma this parallelism is changed, for in this disease the excretion of nitrogen is greatly increased and altogether disproportionate to the introduction of nitrogen. It has not yet been determined to what this is due. We are justified in assuming, at least, that carcinomata exercise a toxic influence, and that the integrity of the cells is thereby destroyed, so that an increased nitrogen excretion results.

In the earlier stages of the disease the patients frequently present a very healthy appearance; the general nutrition, too, may be unimpaired. We should never, however, exclude carcinoma for the reason that the patients look well, are well nourished, or are young. The duration of the disease, however, is decisive. Every case of carcinoma will look relatively well in the initial stages, and be emaciated, weak, and languid in the later stages; a healthy appearance and good general nutrition, therefore, do not *per se* speak against carcinoma excepting in those cases in which the disease has existed for one or several years. In the latter class of cases we are certainly justified in assuming that the disease was not carcinoma from the beginning; we may, however, be dealing with a secondary carcinoma following some other disease of the stomach, or with a secondary carcinoma that is in the developmental stage.

In cases in which some of the signs of carcinoma are present and in which the history shows that the stomach symptoms are relatively recent, and in which, finally, emaciation and cachexia have developed rapidly since the onset of these stomach symptoms, we must seriously consider the diagnosis of carcinoma. At the same time all this is quite frequently seen in other diseases of the stomach, and even the most experienced observer may occasionally be deceived if he attaches too much importance to these symptoms. I have repeatedly encountered patients with disease of the stomach who presented a very cachectic appearance, so that the diagnosis of carcinoma was made. On careful examination, however, it was found that carcinoma was not present. I remember, for instance, a patient who was sent to me with the diagnosis "cancer of the pylorus; secondary ectasy." The patient was much emaciated, very cachectic, vomited a great deal, and was suffering from an advanced degree of ectasy. To the right, under the costal arch, was a small movable tumor that I felt justified in localizing in the pylorus. The stomach-contents was aspirated repeatedly. On each occasion exceedingly large quantities of food-remnants, consisting chiefly of starchy material, were evacuated. This case was examined in 1881 —that is, at a time when examination of the stomach-contents for diagnostic purposes was not universally employed and when the term hypersecretion was completely unknown. The only investigations on ectasy that were known at that time were those of von der Velden, who reported that in one group of ectasies the color-reactions for hydrochloric acid were positive, in another not, and that the latter finding was characteristic for carcinoma.

Contrary to our expectations, the stomach-contents of this patient gave positive color-reactions. Notwithstanding the presence of a tumor and the existence of cachexia, we decided that this was a benign case, basing our opinion chiefly on the color-reaction. As a matter of fact, the patient recovered rapidly. I am unable to say to this day what the significance of the tumor was; all that can be said is that it was not a carcinoma, for the patient gained more than ten kilos in a few weeks, and returned to the clinic four years later in perfect health and looking very well.

It happened that, on the same day, a second patient was admitted to the clinic, who presented an analogous symptom-complex and was also afflicted with an advanced degree of ectasy. In this case, however, no tumor could be felt; the patient, too, was emaciated, but much less cachectic than the first one. I did not doubt that this case was suffering from benign ectasy, just as I was convinced in the case with the tumor, before I examined the stomach-contents, that this patient was afflicted with carcinoma. Both cases were demonstrated together in the clinic. I expected that the analysis of the stomach-contents in the first case would reveal the absence of color-reactions and that in the latter the color-reactions would be positive. Exactly the contrary was found. The first patient recovered, notwithstanding the presence of a tumor and of advanced cachexia; the latter continued to decline and died in a short time. On autopsy the diagnosis of carcinoma was corroborated. These two cases show that cachexia *per se* is no reliable criterion; it is of diagnostic importance only in those cases where it develops very rapidly, and where the quantity and the character of the food and all other factors are carefully considered at the same time.

What has been said in regard to the loss of strength applies also to the weight. It is rarely possible to increase the weight of a cancer case; if it can be done at all, the increase is transitory. In general these patients rapidly lose flesh. Müller examined a case in Gerhardt's clinic and could determine that he lost from 500 to 600 gm. daily.

In advanced stages of the disease the adipose layer is usually completely lost, the muscles are thin and flaccid, and the skin is pale and dry. The latter can usually be raised in high folds that disappear slowly. In exceptional cases hydrops develops.

The objective local symptoms of carcinoma of the stomach are much more important than all the symptoms we have enumerated.

I need hardly mention that cases in which all symptoms point to some disease of the stomach must be submitted to a general examination, and that it is wrong to limit the examination to the diseased organ alone; the state of the organs of the chest—in fact, of all the other organs—should always be investigated carefully. As a matter of fact, it is impossible, for instance, to estimate the size and position of the stomach without carefully studying the position of the diaphragm, the size of the liver, and numerous other factors. I merely mention this one point as an example, but cannot enter into a detailed discussion in this place.

The tongue in carcinoma is, as a rule, thickly coated and covered with a thick, frequently tough, mucus; the taste, therefore, is, as a rule, perverted. It may be stale, pasty, or bitter. One of the most positive signs of the perversion of taste is the aversion that habitual smokers acquire for tobacco. The tongue is rarely found to be clean in carcinoma cases, but is almost always coated.

Swelling of the glands of the neck is not frequent. Swelling of the supraclavicular glands, particularly of the left side, has been credited with some diagnostic importance. The glands in the left supraclavicular region are, however, rarely enlarged. Lépine observed such glandular enlargements only three times in 40 cases of carcinoma. My own observations also show that this complication is rare. If glandular swelling occurs, it usually appears late in the disease. The case of carcinoma of the stomach that Lépine reported in which swelling of the supraclavicular glands appeared before stomach symptoms developed must be considered a rare exception. The patient was a man of forty-eight years, in whom a tumor of the glands of the neck developed about five months before the first attack of vomiting. In this patient digestion had been very good until this attack. The supraclavicular tumor grew to be as large as a hen's egg, and still no tumor of the stomach could be discovered. On autopsy a carcinoma of the pylorus, extending to the head of the pancreas and involving many glands in the vicinity, was found.

Swelling of the glands of the neck is certainly absent in many cases, so that the absence of this symptom in no wise militates against the diagnosis of carcinoma.

Inspection of the abdomen frequently yields valuable clues. In general practice the value of this method of examination is frequently underestimated. In order to recognize visible changes, the region of the stomach should be examined carefully for a long time while the abdominal walls are completely relaxed. In earlier stages of the disease, while the general nutrition is good and a large layer of fat is present in the abdominal wall, the result is frequently negative. In advanced stages of the disease, however, a circumscribed protrusion of the gastric region, corresponding to the tumor, can quite frequently be seen through the thin and relaxed abdominal walls. In cases of advanced ectasy the lower boundary of the stomach can even be seen; it appears as an arched line that is situated somewhere below the umbilicus. If the stomach is situated very low down in the abdomen, the upper boundary of the organ may even be seen. When the stomach is filled, the whole gastric region may protrude. If the patient is instructed to drink the effervescent mixture that we have described, or if the stomach is inflated with air, the appearance presented is much more marked.

If there is gastroptosis,—and this is a frequent complication of carcinoma,—tumors of the lesser curvature may occasionally be seen. If the patients are instructed to inspire deeply, the tumor may be seen to describe distinct respiratory movements. In cases in which narrowing of the pyloric orifice leads to advanced degrees of ectasy, so that the stomach can get rid of its contents only with difficulty, the increased peristaltic movements of the gastric musculature may occasionally be seen through the abdominal walls.

We see, therefore, that inspection alone can occasionally reveal the presence of a tumor. This mode of examination may teach us where the swelling is located, whether it is movable, and in what direction it moves. It may show us that the stomach is dilated or dislocated, or it may lead us to suspect some obstruction in the pyloric orifice of the stomach whenever we see visibly increased peristaltic movements in cases of ectasy. All the information we gain from inspection is, of course, more or less general, and other methods of examination must be carried out in order to amplify and corroborate the conclusions drawn from inspection alone.

Percussion is less important than inspection or palpation. We rarely gain any new information from percussion. If the stomach is small, empty, and contracted, we are altogether unable to determine its boundaries by the most careful percussion as long as the organ is situated in its normal place. If the stomach is ectatic and filled with food, we may occasionally determine its boundaries by percussion, particularly if we examine the patient in different positions. This method, however, is not quite reliable and may lead to error. The form and boundaries of the stomach can be determined with much greater certainty by inflation with air or by the generation of gas within the stomach. Percussion of the tumor usually elicits a dull, tympanitic sound, but rarely teaches us anything that we cannot discover by other methods of examination.

Palpation is more important, for by this method of examination we can determine the consistency, position, size, passive movements, respiratory motility, painfulness, and other peculiarities of the tumor, and at the same time the size, position, and outline of the stomach as far as that is possible.

It is frequently difficult to perform palpation because the patients will not relax the abdomen. Skutsch [1] instructs the patient to place his own hand on the abdomen and to press the abdominal walls inward; in this way he teaches patients how to relax the abdomen. I consider this very practical advice.

If a tumor is present or is suspected, and if we desire to gain some information in regard to the neoplasm by palpation, the patients should first be examined in the horizontal position with the knees drawn up. It is also important to examine the patient while the stomach is filled to different degrees. In order to do this in one sitting the patient should be examined before and after inflation of the stomach. The only way in which to determine with certainty where the tumor is located and that it belongs to the stomach is to examine the stomach when it is distended and filled to different degrees.

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[1] Skutsch, "Die Palpation der Bauch- und Beckenorgane," *Samml. klin. Vorträge*, 1892, No. 48.

However important the presence of a tumor may be in establishing the diagnosis carcinoma of the stomach, we may still err if we reach a conclusion from the presence of a tumor alone. The diagnosis, of course, will be strengthened if other symptoms that indicate carcinoma of the stomach—as the age of the patient, cachexia, rapid emaciation, and loss of strength—are present.

Tumors of the stomach may be situated in different parts of the organ. They are most frequently found in the pyloric region; they may, however, be situated in any other place, or may involve the whole organ. If we feel a tumor in the region of the pylorus, and if there are at the same time ectasy and motor insufficiency of the stomach, and if, finally, the boundaries of the dilated stomach can be pursued as far as the pyloric region, the diagnosis of a tumor of the pylorus is established. Whether or not it is a carcinoma must be determined by subsequent examinations. It is much more difficult to recognize a tumor of the posterior wall of the stomach. If the abdominal walls are thin and the stomach is empty or only slightly filled, it is occasionally possible to feel a tumor in this location; if the stomach is very full or artificially inflated, the tumor can no longer be felt. This symptom may be utilized in diagnosing a tumor of the posterior wall.

Tumors are occasionally found underneath the xiphoid process corresponding to the location of the lesser curvature. If the stomach is in its normal position, they can be felt only during forced inspiration; if the whole stomach, however, is situated low down in the abdomen, they can usually be felt without difficulty. Sometimes they do not appear in this location until the stomach is filled; in other cases, again, they can no longer be palpated when the stomach is filled. This is largely determined by the position of the tumor—that is, whether it is situated nearer the anterior surface of the lesser curvature or nearer to its upper concave margin. In the latter instance distention of the stomach will cause arching of the organ, so that the lesser curvature is forced backward and becomes inaccessible to palpation.

The question of respiratory motility of tumors has been frequently discussed. The majority of authors are inclined to the opinion that tumors of the stomach, in contradistinction to tumors of the liver, do not move with respiration. Leube says that respiration, as a rule, does not exercise any influence on the position of the tumor, but that tumors of the stomach, even if they are not adherent, occasionally move with the diaphragm, the liver, and the spleen.

It can easily be determined that the stomach, when it is situated in its normal position, even when it is ectatic, possesses respiratory motility.

This has been corroborated by transillumination. If there is gastroptosis, this motility seems to be lost. It is true that the stomach occasionally performs small respiratory excursions, even if there is gastroptosis, and that tumors of the organ follow these movements. This occurs, however, only in cases of partial ptosis. In vertical position of the stomach respiratory motility may be seen if the stomach in its totality is dislocated downward, but this motility remains very slight or is lost chiefly because the stomach is no longer in contact with the diaphragm. Respiratory motility, however, as we have said above, may persist even though there be advanced ectasy.

In general, tumors of the curvatures of the stomach show greater respiratory motility than tumors of the pylorus. If tumors of the pylorus, however, are adherent to the liver, they follow the excursions of the diaphragm together with the liver. The type of breathing must always be considered when estimating the significance of respiratory motility of a tumor of this kind. Many subjects, particularly if they are requested to take a deep breath, are in the habit of performing costal respiration—that is, they dilate the thorax so that the epigastrium, instead of being pushed out, is drawn in. This is seen not only in men, but quite frequently in women, and is particularly common if there is pain in the abdomen. We must always consider this factor in testing the respiratory motility of tumors. The presence or absence of respiratory motility is important only if we consider all these factors and study them in connection with other symptoms.

Normally, the lesser curvature of the stomach is not in contact with the anterior abdominal wall, but is covered by the margin of the liver. For this reason small tumors of the lesser curvature may not be palpable; if the whole stomach, however, is dislocated downward or if it is placed vertically, tumors of the lesser curvature can be palpated. If the abdominal walls are relaxed and the patients are very thin, the pancreas may occasionally simulate a tumor of the lesser curvature, particularly if the stomach is slightly dislocated downward. As a matter of fact, the pancreas can occasionally be felt through the stomach when it is empty, even though the latter is not dislocated downward. I have repeatedly encountered cases in which the pancreas was erroneously taken for a neoplasm of the stomach. If the patients are repeatedly examined with care and if the stomach is artificially inflated, such an error can usually be avoided.

Many tumors of the stomach are palpable only when the patients occupy certain positions.

In the year 1884 a gentleman of thirty-three years was under my care who had been suffering from stomach trouble since his fifteenth year. During a certain period in the past his distress had been relieved and all the stomach symptoms had disappeared, with the exception of a certain weakness of the stomach. In 1888 severe stomach symptoms reappeared that corresponded to the symptoms of ulcer. This period was again followed by a prolonged respite from distress. In the beginning of 1894 pain returned, but this time the character of the pain was different: it was more persistent, more irregular, and seemed to be less dependent on the quality and the quantity of the food. Within the last five months the patient had lost twenty-five pounds. An examination in the dorsal position failed to reveal the presence of a tumor; if, however, the patient was placed on the right side and instructed to inspire deeply, a hard, slightly nodular tumor that was painful on pressure could be felt in the median line between the umbilicus and the ensiform process. No ectasy of the stomach was found.

The stomach-contents was repeatedly examined; no residue was found, the values for hydrochloric acid were slightly increased, and there was no lactic acid. The diagnosis, carcinoma on the basis of an old ulcer, was made, and an exploratory laparotomy proposed. This operation was performed, and it was found that the mesentery was filled with a number of carcinomatous nodules that varied in size from a pea to a bean. When the finger was introduced into the peritoneal cavity a tumor of the stomach could be felt that was adherent to the diaphragm. The operation, of course, could not be performed, owing to the presence of metastases and adhesions.

Although no autopsy was made in this case, the diagnosis of carcinoma may be considered established. It was remarkable that the tumor could be felt only when the patient was lying on the right side, and that it appeared much smaller on palpation than it was found to be when examined through the laparotomy wound. My experience does not coincide with that of Ewald, for the latter author claims that most tumors feel larger to the palpating finger than they are in reality. As a matter of fact, I have frequently observed the opposite. I need hardly mention that the presence of severe ascites renders the discovery of a tumor of the stomach impossible.

It is also frequently difficult to differentiate tumors of the lower margin of the liver, particularly tumors of the gall-bladder, from tumors of the stomach. In these cases inflation of the stomach is of paramount importance. If a tumor that seems to be situated in close contact with the margin of the liver moves away from this position during inflation of the stomach, or if it moves laterally and it can be determined that it merges directly into the boundaries of the stomach, the diagnosis of tumor of the stomach may be made.

Inflation.—I have repeatedly insisted that the stomach should always be examined both when it is empty and when it is distended, in case a tumor is found in the gastric region. In order to do this we employ artificial inflation.

The first thing to observe is whether or not the tumor remains in the same position and maintains the same form before and after the stomach is inflated. Occasionally it will be found that it changes its position or that it becomes better palpable or less readily palpable during inflation, or, finally, that it can no longer be felt after the stomach is distended with air. As a rule, particularly in tumors of the pylorus, it is an easy matter to show by inflation that the tumor is connected with the stomach. Tumors of the pylorus generally move to the right during inflation—quite frequently slightly downward, less frequently upward. Tumors of the posterior wall that are palpable before inflation are frequently no longer palpable after the stomach is inflated. The same applies to tumors of the lesser curvature.

If the tumor is freely movable and if it changes its position during inflation, this may be considered a proof that no solid adhesions with neighboring organs exist. I consider this question of importance, particularly when operative interference is being considered. If it is possible to lift the tumor away from the liver by inflation, and if we can palpate its upper margin after this is done, tumors of the liver and of the gall-bladder can usually be excluded.

Under certain circumstances the pyloric orifice closes so that nothing can pass from the stomach into the intestine. There are, however, cases in which it can no longer close completely; this condition has been called by Ebstein incontinence of the pylorus. If an effervescent powder is given to a healthy person, the whole stomach becomes inflated; this is due to the fact that the pylorus contracts at once and closes the orifice completely. After a time, however, it allows the gas to escape into the duodenum. If the pylorus is incontinent, the stomach is not inflated to such a degree because a part of the gas that is developed immediately escapes into the intestine. According to Ebstein, this incontinence of the pylorus is a symptom that is observed in a variety of lesions of this part of the stomach, particularly in ulcerative destruction and in infiltration of the pylorus. If the presence of a carcinoma of the stomach can be established, incontinence of the pylorus indicates that the growth is located in this portion of the stomach.

Some authorities claim that insufficiency of the pylorus is a purely physiologic phenomenon. I do not, however, agree with this view. I am in the habit of performing inflation of the stomach in nearly all the stomach patients whom I see, and am in a position to state that normally the pylorus closes completely as soon as carbon dioxid gas develops in the stomach. Some time always elapses before the gas escapes, and then it usually passes out through the cardia before it passes through the pylorus. Insufficiency of the pylorus is unquestionably pathologic; it is also certain that this symptom is occasionally seen in carcinoma of the pylorus; but it is likewise true that it is frequently absent.

I wish briefly to call attention to two other methods of examination that aid the diagnosis in many cases—namely, high rectal injections (for the purpose of removing the bowel-contents) and artificial inflation of the colon. If these two methods are employed, it is usually an easy matter to exclude tumors of the transverse colon or fecal tumors in this part of the intestine. In doubtful cases artificial inflation of the colon by air is a very valuable adjuvant to the diagnosis, because it aids us in discovering the position of the tumor and in determining the organ to which it belongs.

I am hardly in favor of the method of electric transillumination that some authors recommend in the diagnosis of tumors of the stomach. In the first place, this method is difficult to carry out; in the second place, many other lesions, as peritoneal thickening, dislocation of neighboring organs, etc., may oppose the passage of light in the same way as tumors of the stomach, so that even a positive result does not enable us to diagnose the presence of a tumor. Transillumination with Röntgen rays has so far given no positive results. [Transillumination of the stomach by Einhorn's method has not been found difficult to execute, and the results obtained are sometimes useful.—Ed.]

If the carcinoma is situated in the cardia, the symptoms are slightly different from those we have described. Dilatation of the stomach, of course, never occurs in carcinoma of the cardia, whereas it is the rule in carcinoma of the pylorus. The symptoms of the two conditions vary in other respects; in the first place the patients usually complain of distress immediately after eating. Other subjective symptoms are a feeling of pressure and discomfort, or even of pain in the epigastric region. All these symptoms frequently appear as soon as anything is swallowed.

There are, however, a great many cases in which symptoms of this character are absent for a long time; the only complaints of such patients may be slight loss of appetite and increasing weakness. Nothing whatever indicates the development of a malignant neoplasm in the cardia. If a stomach-tube is passed, we shall frequently be surprised to find an obstacle at the cardia. In the beginning this obstacle, of course, is inconsiderable, so that even comparatively large sounds can be inserted into the stomach. In other cases, again, the patients complain of a feeling of pressure in the cardiac region very early in the course of the disease. Occasionally the location of this feeling of pressure is described as higher up or sometimes as lower down. The patients usually notice that solid food seems to stick, but that fluid or pultaceous material can be swallowed without difficulty. As the stenosis of the cardiac orifice of the stomach increases, these symptoms exacerbate; the pain appears at all times, and is no longer dependent on swallowing. In addition, vomiting or regurgitation of mucus occurs. Later in the course of the disease a diverticulum forms above the stenotic portion of the stomach, in which a portion of the ingesta is apt to accumulate. This leads to irritation of the mucosa and increased secretion.

The most important objective symptom of carcinoma of the cardia is the obstruction of the cardiac orifice. In order to determine the existence of this stenosis sounds of different caliber must be passed and the width of the orifice determined by the caliber of the largest sound that can pass through. Occasionally we find that the result of this examination does not correspond with the difficulty that the patients experience in swallowing. The patients, for instance, may be able to swallow finely divided food without difficulty, and at the same time it may be impossible to insert even a very thin sound into the stomach. In cases of this kind we are justified in assuming that the stenosed canal is not straight, but that it has become curved by the development of the carcinoma, so that fine particles of food can pass through, whereas a straight sound is arrested.

The examination with the sound merely tells us that an obstruction exists in the cardia, but does not teach us whether or not this obstruction is carcinomatous. In order to determine the latter point all the other symptoms of the case must be carefully investigated, the course of the disease must be studied, and the age of the patient considered. On the other hand, all other possible causes of stenosis must be excluded. It is always best at first to pass a soft stomach-tube instead of a sound.

Boas succeeded in one case in diagnosing cancer from small characteristic tumor particles that he removed with the sound. This, of course, was an exceptional occurrence. Mucus mixed with blood is, however, quite frequently found in the openings of the sound.

Less significant than all these symptoms are pain on percussing the xiphoid process and the absence or the retardation of the second swallowing sound ("Druckpressgeraüsch"). It is worth while, however, to examine the patient for these symptoms. The most important sign, after all, is stenosis of the cardia, and as long as this symptom is absent the diagnosis of carcinoma of the cardia can never positively be made.

After this brief discussion of the specific symptoms of carcinoma of the cardia we will continue our general description of the systematic examination of the stomach in carcinoma. After having discovered what we can by inspection, percussion, and inflation of the stomach, we proceed to the examination of the specific powers of the organ—namely, its secretory, motor, and absorptive functions. We have already mentioned that von der Velden, in 1879, stated that in stenosis of the pylorus due to carcinoma free hydrochloric acid is absent from the stomach-contents. I might mention that von der Velden did not claim that this absence of free hydrochloric acid was a pathognomonic sign of carcinoma of the pylorus. He stated expressly that the absence of free hydrochloric acid may also be due to gastritis or to fever.

Ewald has called attention to the fact that Bird, as early as 1842, studied the hydrochloric acid and the organic acids in a case of dilatation of the stomach combined with carcinoma of the pylorus. He performed a series of analyses of the vomit, and found that "free hydrochloric acid is present in the vomit during the irritative stage of the disease, but gradually decreases in proportion to the loss of strength; the organic acids, however, seem to increase as the free hydrochloric acid decreases." I do not think that this old and isolated observation of Bird, which remained completely unnoticed, impairs the credit that is due to von der Velden for studying the question with new methods and in a relatively large number of cases.

No question has probably been so much discussed within the last decade as the hydrochloric-acid findings in carcinoma of the stomach and their diagnostic significance. Some authors are inclined to consider the absence of hydrochloric-acid reactions as a pathognomonic sign of carcinoma, while others deny that hydrochloric acid is, as a rule, absent in carcinoma. One of the chief reasons why different authors make such contradictory statements is the fact that they have not distinguished between free and combined hydrochloric acid. It may be considered established to-day that free hydrochloric acid is, as a rule, absent in carcinoma, and that the color-reactions for free hydrochloric acid are usually negative. The controversy that is still being waged to-day regards a subordinate question—namely, whether or not this absence of free hydrochloric acid, or, better, the reduction in the production of hydrochloric acid, can be considered a pathognomonic sign of carcinoma. The discussion of this question, it appears to me, is altogether useless, for von der Velden in his day settled this matter by demonstrating that gastritis or fever may also lead to the disappearance of free hydrochloric acid in the stomach-contents.

The fact, however, that this symptom is observed in a variety of conditions does not impair its value as one of the symptoms of carcinoma, although, of course, it cannot be considered pathognomonic in the proper sense of the word. I have always emphasized the great diagnostic importance of this symptom, and have based my view on experiments that I began immediately after von der Velden published his results and have continued ever since. Very soon after I began my investigations I called attention to the fact that free hydrochloric acid may be absent in different diseases of the stomach. It was in my clinic that the absence of free hydrochloric acid was first established in amyloid degeneration of the gastric mucosa and in toxic gastritis. I am of the opinion that the value of this symptom in the diagnosis of carcinoma is not impaired by the fact that free hydrochloric acid may be absent permanently or for a time in amyloid degeneration of the stomach, in toxic gastritis, in atrophic catarrh, in certain forms of nervous dyspepsia, and occasionally in phthisis, heart-lesions, etc. I say this because no competent clinician is apt to confound any of these diseases with carcinoma.

I do not wish to convey the impression that the symptom can be utilized in such a manner that the stomach-contents need be aspirated and examined only for free hydrochloric acid, and carcinoma immediately be diagnosed if free hydrochloric acid is absent; the symptom is important only when considered in connection with the whole symptom-complex. I also wish to insist particularly that we should never be content with one analysis of the stomach-contents. Unfortunately, many clinicians are in the habit of doing this. The only way in which to avoid error is to control the analysis by frequent repetition. Carcinoma of the stomach is often complicated with advanced degrees of ectasy, and the only way in which to gain a clear insight into the true condition of the stomach is to analyze the stomach-contents repeatedly.

Other important questions are the following: At what stage of the disease may we expect to find free hydrochloric acid absent in carcinoma of the stomach? Is this absence of free hydrochloric acid an early or a late symptom of the disease? These questions are intimately connected with another one—namely, What causes the reduction in the secretion of hydrochloric acid?

When I began my investigations I was inclined to the opinion that the carcinoma itself, or possibly certain products that the neoplasm generated, destroyed the hydrochloric acid of the stomach. This view has since been shown to be erroneous. Nowadays it is almost universally conceded that the gastritic process that accompanies carcinoma and that leads to atrophy of the gastric mucosa must be made responsible for the loss of hydrochloric-acid secretion. As a matter of fact, it is easy to show that the production of hydrochloric acid decreases in proportion to the development of the carcinoma. In many cases, however, this symptom is discovered in early stages of the disease. In the first paragraphs of this section I reported 2 cases in which the dyspeptic symptoms were relatively slight and of short duration, in which there was no tumor, no ectasy, no emaciation, no cachexia, and in which, nevertheless, free hydrochloric acid was constantly absent. Cases of this kind are not rare by any means. To judge from my personal experience, I am inclined to say that the absence of free hydrochloric acid is an early symptom of carcinoma of the stomach. There are, however, exceptions to this rule; long ago I reported 2 cases of this kind, one of which I will quote as an example:

The patient was a miner of sixty-three years. He had been perfectly healthy all his life. In February, 1890, he began to complain of loss of appetite and pain in the region of the stomach, combined with frequent belching. The patient entered the clinic in June. On examination we found a poorly nourished, weakly individual with a distinct tumor in the epigastric region, advanced ectasy, and cachexia. Up to within two weeks before his death free hydrochloric acid could be discovered in the stomach-contents, and it could be determined that the peptic powers of the stomach were still good.

On autopsy the diagnosis of carcinoma of the pylorus with dilatation of the stomach could be corroborated; in addition, metastatic carcinomata of the portal and retroperitoneal glands were found. I never was able to explain why in this case of cancer the secretion of gastric juice was so abnormal, so that I am inclined to consider the case a rarity. There is another group of carcinomata, however, in which free hydrochloric acid, even hyperacidity, is found for a long time. These are the cases in which the cancer develops on the basis of an ulcer, for the latter lesion, as I have demonstrated above, is, as a rule, though not always, complicated with hyperacidity. We can readily understand, therefore, why in such cases, particularly when the carcinoma is just developing, hyperacidity is frequently found.

Thiersch was the first to report an observation of this character in 1885. He examined all the cases of carcinoma that entered Wagner's clinic, and found that the methyl-violet reaction for free hydrochloric acid—in vogue at that time—was negative in all cases excepting the following one, which he reported as a rarity. This patient was a man of only twenty-six years, who had been suffering from gastric distress and frequent vomiting for a year. On several occasions blood was vomited. Several times the stools were black. The stomach-contents always gave a distinct methyl-violet reaction. The diagnosis ulcer of the stomach was made. On autopsy it was found that the pylorus was very much stenosed; immediately above it a cicatrized, kettle-shaped ulcer of 5 cm. in diameter was discovered. Numerous carcinomatous nodules were found in the liver. On microscopic examination nests of cylindric epithelium were found disseminated throughout the margin of the ulcer and the musculature of the pylorus. These nests were scattered through these tissues at varying depth, so that this finding corresponded to the combination of gastric ulcer and carcinoma that Hauser first described. Thiersch regarded the case as incipient carcinoma following ulcer.

Kruckenberg, in 1888, described another case of carcinoma that developed on an ulcer that was undergoing cicatrization. Here the tests for free hydrochloric acid were positive in one-half of the analyses of stomach-contents that were made. We are indebted to Rosenheim for the most exhaustive investigation into this question. He calculated that at least 6 per cent. of all cases of carcinoma of the stomach develop from ulcer. Such cases are characterized by normal gastric secretion that may continue until death ; they are distinguished by this symptom from the majority of cases of ordinary carcinoma of the stomach.

Rosenheim explains this phenomenon by assuming that the carcinoma develops chiefly in the deeper portions of the ulcer, so that the mucous membrane of the stomach remains intact for a long time.

There are, however, exceptional cases in which the secretion of hydrochloric acid remains unimpaired for a long time, even though no ulcer precedes the carcinoma. I have already mentioned this above, and have given an example of such a case. It has been shown by numerous analyses of the stomach-contents that the production of hydrochloric acid is rarely completely arrested, even in those cases of carcinoma in which free hydrochloric acid is absent. More or less combined hydrochloric acid can usually be found in the majority of cases. Ewald calls particular attention to the fact that cases in which the secretion of hydrochloric acid is completely lost are exceedingly rare. We possess two methods for determining the intensity of the secretory perversion in cases where free hydrochloric acid is absent: the one is to determine the quantity of combined hydrochloric acid ; the other is to determine the hydrochloric acid deficit. The latter method is the simpler and the more practical one of the two. One-tenth normal hydrochloric acid solution is added to the filtrate of the stomach-contents until a distinct Congo or phlorogluein-vanillin reaction occurs. This method enables us to gain a much more reliable idea of the deficit than a determination of the combined hydrochloric acid, however exact the latter may be. If the deficit is determined from time to time, we can easily control the progress of the disease and draw definite conclusions in regard to the development of atrophy of the secretory apparatus of the stomach.

We may expect, *a priori*, that the secretion of pepsin will be reduced in those cases of carcinoma in which the secretion of hydrochloric acid is greatly reduced. Stomach-contents from cases of this kind do not, as a rule, digest a disc of albumin, even though a sufficient quantity of hydrochloric acid is added.

Hammerschlag found the pepsin secretion very much reduced in nearly two-thirds of the cases of carcinoma that he examined. This lack of pepsin, however, is no specific symptom of carcinoma, for the cancer itself does not influence the secretion of pepsin, of hydrochloric acid, nor of rennet. All these perversions of secretion are due merely to secondary gastritis or atrophy of the gastric mucosa. The more advanced the latter conditions, the more will the secretion of ferments be reduced. Boas and Oppler, in some cases of carcinoma in which the carcinoma was removed by resection of the pylorus, saw an increase in the secretion of hydrochloric acid and of pepsin. This can be due only to the fact that removal of the carcinoma also stopped the gastritic process, so that those portions of the secreting parenchyma that were not completely destroyed could recover.

The secretion of rennet-ferment and rennet-zymogen is decreased in carcinoma of the stomach as in any other process that leads to atrophy of the gastric mucosa.

In order to control the analysis of the stomach-contents it is well to perform an artificial digestion with gastric juice, with and without the addition of hydrochloric acid. Rosenbach, it is true, argues that this test is useless, because we do not care to determine whether the gastric juice removed from the stomach possesses digestive powers, but merely wish to know whether the gastric juice can perform its normal functions while it is still in the stomach. I do not agree with this view. He is wrong, above all, when he advises examining the filtrate for peptone and when he claims to be able to determine the digestive powers of the gastric juice within the stomach by determining the presence or absence of peptone in the aspirated stomach-contents. In carcinoma the peptone reaction is nearly always positive, but this does not demonstrate by any means that the production of gastric juice is sufficient. The normal stomach always produces more gastric juice than is needed for the digestion of albumin, and for this reason we always find free hydrochloric acid at a certain stage of digestion. The peptone reaction can never teach us whether all the proteid material is combined with hydrochloric acid ; the only way to determine this is to calculate the hydrochloric-acid deficit. As a rule, macroscopic inspection of the stomach-contents alone tells us all we want to know, for the presence of numerous coarse, undigested particles of meat shows that the digestion of albumin is insufficient, even if the peptone reaction is positive in the filtrate of the stomach-contents.

We must remember, therefore, that in carcinoma of the stomach insufficient production of hydrochloric acid is the rule. This symptom, as we have said, is not pathognomonic for carcinoma, but it possesses a certain diagnostic importance when considered together with other symptoms.

Of late years Boas, in particular, has taught us to attach diagnostic significance to the presence of lactic acid in the stomach. Formerly the presence of lactic acid in the stomach during certain stages of digestion was considered normal. Later, Martius and Lüttke demonstrated for the first time that lactic acid is not a normal factor of digestion, and that lactic-acid fermentation, if it is at all considerable, must always be considered a pathologic process. Boas was the first to describe an exact method for the quantitative and qualitative determination of lactic acid, and he, too, arrived at the conclusion that "no lactic acid is produced when carbohydrates are introduced into a healthy stomach"; he further succeeded in demonstrating the presence of considerable quantities of lactic acid in nearly all cases of carcinoma of the stomach. He was careful to establish the same conditions in healthy and in carcinomatous cases when performing this examination. He attaches much importance to the presence of lactic acid in carcinoma of the stomach, and considers it one of the early symptoms of the disease, and goes so far as to claim that the presence of lactic acid enables us to make the diagnosis of carcinoma at a stage when no tumor can be palpated or when the tumor is covered by other organs, particularly the liver.

I am inclined to be less enthusiastic in regard to this matter, possibly because it is not so new to me. The demonstration of the presence of lactic acid appears to me to have a significance analogous to the determination of the absence of free hydrochloric acid. Both conditions are most frequently found in carcinoma, but both may occasionally be absent and both may exceptionally be found in other diseases of the stomach. Ever since I began to perform systematic analyses of the gastric contents in every case of chronic disease of the stomach,—that is, for more than sixteen years,—I have always paid attention both to the presence or absence of hydrochloric acid and the presence or absence of lactic acid. I have known for a long time that lactic acid is present in the majority of cancer cases. One of the chief conditions for the formation of considerable quantities of racemose lactic acid is motor insufficiency.

I have quoted an example of a case of carcinoma of the stomach in which lactic acid was present at a stage of the disease in which no tumor could be felt. I do not think, however, that we are justified in drawing the conclusion from such cases that the presence of lactic acid is necessarily an early symptom of carcinoma. True, it may be an early symptom, but more frequently it is a late symptom. If secretory and motor insufficiency occur early, lactic acid will necessarily be present. We are no more justified in generalizing in regard to lactic acid than we are in regard to the tumor, and in neither instance can we say that lactic acid or a tumor is an early symptom or a late symptom of the disease; for there are tumors that are accessible to palpation very early in the disease, owing to the position they occupy or the portion of the stomach they involve, and, on the other hand, there are cases of carcinoma in which no tumor can be discovered with the methods at our disposal even in the latter stages of the disease.

If we wish to understand the true significance of lactic acid in the stomach, we must determine what conditions favor its appearance. In the light of our present knowledge we can say that two conditions must be fulfilled in order that lactic acid appear: in the first place, the secretion of gastric juice must be greatly reduced; in the second place, there must be stagnation of stomach-contents. A third factor that favors the formation of lactic acid is deficient absorption, which usually accompanies the former two abnormalities. Sticker[1] has demonstrated that the passage of amylaceous material through the mouth almost always leads to the formation of more or less lactic acid. Normally, this lactic acid, like all other soluble products of digestion, is rapidly absorbed; when absorption, however, is rendered difficult; when there are, at the same time, stagnation of stomach-contents and secretory insufficiency—

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[1] *Münch. med. Wochenschr.*, 1896, No. 26.

most favorable conditions for the continuation of this bacterial lactic-acid formation are created in the stomach.

Hammerschlag has recently called attention to certain cases in which free hydrochloric acid was greatly reduced or absent, in which there was stagnation of stomach-contents, but in which, nevertheless, no formation of lactic acid occurred. In these cases, however, the digestion of albumin was normal or only slightly impaired, whereas it was always decreased or completely inhibited in all those cases in which lactic-acid fermentation occurred. He is inclined to the belief, therefore, that the gastric ferments play a certain rôle in the generation of lactic acid.

I agree with Hammerschlag in his statement that lactic-acid fermentation rarely occurs in cases in which the digestion of albumin is normal or only slightly decreased, and that it occurs particularly in those instances where the production of hydrochloric acid is greatly reduced. Whether or not the deficiency of gastric juice is concerned in this matter remains an open question. The absence of free hydrochloric acid is no exact index of the intensity of the secretory perversion; it only demonstrates that the production of hydrochloric acid is quantitatively perverted. It does not indicate how far the production of hydrochloric acid deviates from normal, so that, notwithstanding the absence of free hydrochloric acid, the production of gastric juice may be just sufficient, or it may, of course, be very much reduced.

As a rule, the production of hydrochloric acid and the secretion of pepsin run an approximately parallel course. We should not expect to find, however, that pepsin is absent in all cases where free hydrochloric acid is absent, for the absence of free hydrochloric acid is by no means identical with inhibition of hydrochloric-acid production. It may be considered established that pepsin is, as a rule, absent in all cases where the production of hydrochloric acid is greatly reduced, and as both hydrochloric acid and pepsin disappear from the stomach-contents at the same time, it is certainly more nearly correct to consider a reduction of gastric secretion and not the reduction of hydrochloric acid alone as one of the factors that favor the production of lactic acid. Other factors that also favor this production are stagnation of stomach-contents and deficient absorption.

All these conditions are most frequently fulfilled in carcinoma of the stomach, particularly in carcinoma of the pylorus. The conditions, however, may also be absent in carcinoma of the stomach, or, on the other hand, may be present in diseases of the stomach other than cancer. Recently, for instance, I studied a case of carcinoma of the stomach in which lactic acid was absent. In this case the diagnosis was corroborated by a laparotomy, and it was found that the carcinoma had developed on the basis of an ulcer. The stomach-contents contained free hydrochloric acid, and there was neither ectasy nor motor insufficiency.

Inversely, we frequently obtain a distinct lactic-acid reaction in the absence of carcinoma. This is seen particularly in cases of atrophic catarrh with atony. Rosenheim and others have reported cases of this kind, and any observer who has much clinical material will have observed the same. It is true that the appearance of lactic acid in cases that are not carcinomatous is rare as compared to the frequency with which lactic acid is found in carcinoma of the stomach, but the appearance of lactic acid can, nevertheless, hardly be considered a pathognomonic sign of carcinoma. If lactic-acid fermentation occasionally occurs in isolated cases that are apparently not complicated with atony of the gastric mucosa, this naturally does not disprove the fact that secretory and motor insufficiency of the stomach are the two factors that chiefly favor the formation of lactic acid. Strauss demonstrated in a case that he studied in my clinic that ferment organisms may be present in a stomach with carcinoma and carcinomatous induration, even if only a small residue of coarse ingesta is present.

The only methods for determining the presence of lactic acid that are of practical importance are those that are easy of execution. The simplest test is the one with Uffelmann's reagent, as modified by Kelling. Strauss has also recommended a modification[1] that I can indorse and that I have found to be exact. The test for lactic acid should be performed on stomach-contents that is removed early in the morning before breakfast. If the stomach is found empty at this time, this alone indicates that there is no advanced degree of stagnation. The test, of course, can also be performed with stomach-contents that is aspirated after a test-meal. The stomach-contents removed after a test-breakfast is less suitable.

In view of the fact that more or less abundant quantities of lactic acid are introduced into the stomach with the food, Boas has recommended a test-meal containing no lactic acid whatever. This is a rational procedure, because we do not alone wish to determine whether lactic acid is present in the stomach, but merely whether it is formed there. Boas's test-meal consists of soup made of Knorr's oatmeal (without milk or butter); the stomach is first washed out and then this meal administered. In practice this modification is not necessary, and an ordinary meal may be administered, for the quantity of lactic acid that is normally introduced with an ordinary test-meal is not sufficient to give a positive Uffelmann reaction.

We see, therefore, that lactic-acid fermentation is not absolutely pathognomonic for carcinoma. It is true, on the other hand, that lactic-acid fermentation occurs most frequently in carcinoma, and that the presence of lactic acid is, therefore, of great diagnostic importance. We could consider lactic acid a pathognomonic sign of carcinoma only if it were a specific product of this form of neoplasm, but this is not the case. It is simply a product of a variety of lactic-acid-forming micro-organism that vegetates on the carbohydrates of the food in the stomach. The most interesting of these bacterial species are the long, thread-like bacilli that Boas and Oppler described for the first time. These bacilli are almost constantly present in the stagnating stomach-contents of cancer cases. It has been established that they can form lactic acid, but it has also been determined that they are not the only species that can do this. At the same time they possess a certain diagnostic value, for they are usually present in large numbers wherever abundant quantities of lactic acid are found in cases of carcinoma of the stomach. The discovery of isolated specimens of these bacilli possesses no significance.

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[1] Compare p. 117.

These are the most important points in regard to the chemism of the stomach-contents in carcinoma. The examination of aspirated stomach-contents, however, should not be limited to these methods. It should always include macroscopic inspection, like the examination of any other stomach-contents.

As a rule, macroscopic examination of the aspirated stomach-contents reveals certain peculiarities. If the stomach-contents is removed five or six hours after a test-meal, abundant quantities of coarse food-particles, in particular coarse particles of meat, but also coarse remnants of bread, potato, etc., are found. Not only the proteolytic, but also the amylolytic, power of the stomach is disturbed. The quantity of residue that is found depends largely on the motor power of the stomach; the more this is impaired, the greater the amount of residue. If, on the other hand, the motor power remains intact, the ingesta may be propelled into the intestine in a normal manner, even though the peptic power of the stomach is greatly reduced. If, therefore, the stomach is found empty from four to six hours after a test-meal, this does not by any means demonstrate that the digestive powers of the organ are normal and good; it merely demonstrates that the motor power is good. The only way in which to gain an insight into the peptic powers of the stomach is to analyze the stomach-contents. If lavage of the stomach, performed five or six hours after a test-meal, reveals the absence of stomach-contents,—and this is quite frequently the case in early stages of the disease,—the stomach should be washed out again, but sooner after a test-meal. If, on the other hand, very abundant food-particles are found six hours after a meal, and if the remnants are coarse and barely digested; if, at the same time, free hydrochloric acid is absent and the digestive powers of the stomach are found reduced, we may know that both the secretory and the motor functions of the stomach are reduced.

From a practical point of view it is very important to determine both the secretory and the motor powers of the stomach. If the former alone are disturbed, the outlook is undoubtedly more favorable. A perversion of the secretory function alone is quite frequently found in the earlier stages of carcinoma, particularly in those cases in which the carcinoma is not situated in the pylorus, or in which at least no stenosis of this part of the stomach has been produced. I agree with Schüle that the motor power of the stomach is not always reduced in carcinoma of the pyloric region, even in the earlier stages. Many authors seem to believe that carcinoma of this region leads to serious disturbances of the motor function very early in its development, so that stagnating stomach-contents can always be found at a time when the stomach should be empty. Schüle examined 53 cases of carcinoma of the pylorus and found a retardation of motor function in only 7 cases—that is, in 13 per cent. The exact proportion of cases in which this occurs will naturally vary in different series; at the same time it may be regarded as established that the motor power of the stomach may be good in early stages of carcinoma, even though the secretory power of the stomach is greatly reduced at the same time. The motility of the stomach may also be reduced, even if there is no stenosis of the pylorus. This, however, does not frequently occur.

Strauss[1] has recently called attention to another sign of carcinoma. In 5 cases of carcinoma that were not situated in the pylorus, but in the lesser curvature or in the fundus, he found that fermentation was relatively abundant in the stomach-contents that was removed from these cases and immediately placed in the incubator in a fermentation tube; he says, relatively increased, as compared to the quantity of residue found after a test-breakfast. This author is inclined to consider this contrast between good motility and increased fermentation a diagnostic criterion in doubtful cases, and believes that it indicates that the surface of the mucosa is mechanically roughened; in other words, he explains the apparent contrast between normal motility and increased fermentation by assuming that the ridges and furrows formed in the gastric mucosa form a welcome retreat for microparasites. At the same time the motility of the stomach, he argues, need not be perverted.

Pronounced yeast fermentation is rarely observed in carcinoma. Isolated yeast-cells are occasionally found, but an abundant development of gas is rarely observed.

The stomach-contents in these cases, like the vomit, frequently contains black coffee-ground-like material that is derived from blood. This is quite frequently found in cases in which no evidence of hemorrhage into the stomach existed.

This practically includes everything that we can learn from analysis of the stomach-contents that is removed by aspiration. We see that we can do more than merely determine the quantity of hydrochloric acid: the determination of the quality, the color, and the general appearance of food-remnants, the examination for lactic acid, etc., are just as important, if not more so. I believe that the analysis of stomach-contents would have been introduced into practice and would have been appreciated much sooner if so many investigators had not limited their attention for so long a time to hydrochloric acid alone, and had not devoted all their energies to discovering very fine and ultrafine methods for determining minimal traces of hydrochloric acid.

Microscopic examination should be performed first with stomach-contents that is aspirated, second with vomit, third with the wash-water. Characteristic particles are rarely found in this material. A concentrically arranged collection of cells would be found only in the squamous tumors that start from squamous carcinoma of the esophagus. The tubules and cell-nests of the cylindric-celled carcinoma show no cylindric arrangement. We could make the diagnosis carcinoma from the examination of isolated cell-nests only if we were positive that the tumor came from the stomach. If whole tumor-particles are aspirated from the stomach, a carcinomatous structure, of course, immediately enables us to render the diagnosis. Rosenbach and Boas have described cases of this kind.

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[1] "Verein f. innere Med. in Berlin," supplement No. 24, in *Deutsch. med. Wochenschr.*, 1896, No. 38.

Reineboth has recently called attention to the fact that in cancer of the stomach large and small coagula of blood are occasionally washed out during lavage, and that small tumor-particles are occasionally found within these coagula. He examined 8 cases of carcinoma and found such coagula in 5. Twice he was able to make the diagnosis carcinoma from the microscopic picture that was presented. It seems, therefore, that we should examine these coagula in the wash-water very carefully. It is probable that characteristic findings will be discovered only in the later stages of carcinoma.

[On the question of the possible early determination of the presence of carcinoma by examination of fragments of the gastric mucosa recovered in the wash-water Hemmeter[1] believes that he has occasionally been successful in making a diagnosis by this method. Most observers regard this criterion as absolutely valueless. Einhorn, in a recent article,[2] suggests that the diagnosis of carcinoma of the stomach may be made in this way under specially favorable conditions if a direct invasion of the gland-substance by epithelial cells can be observed.—Ed.]

The other microscopic constituents of the stomach-contents are not characteristic. We usually find coarse and undigested muscle-fibers, remnants of vegetable material, starch-granules, fat-droplets, and numerous fungi; sarcina ventriculi is rarely found in carcinoma, whereas, as we have seen, it is frequently present in atonic and benign ectasy. Oppler has demonstrated that carcinoma of the stomach is an unfavorable soil for the development of sarcina by introducing pure cultures of sarcina into the stomachs of cancer cases. All the germs disappeared within twenty-four hours. Yeast-cells are quite frequently found in the stagnating stomach-contents of carcinomatous cases, although, as a rule, they are present only as isolated specimens.

The discovery of the long, thread-like bacilli that we have described above is more important (compare Figs. 20 and 21, which illustrate the findings in two different cases); Fig. 20 is from a case of carcinoma that developed on the basis of an ulcer; Fig. 21, from a case of advanced carcinoma of the pylorus.

These thread-like bacilli are almost regularly found in the stagnating stomach-contents of carcinomatous cases. The significance of this finding is not impaired by the fact that the same bacilli are occasionally found in the stomach-contents that contains free hydrochloric acid (Rosenheim and Richter). Schlesinger and Kauffmann succeeded in making pure cultures of these bacilli and in demonstrating that they had no specific importance in carcinoma; at the same time the very fact that they occur with such constancy gives them a certain diagnostic importance. They are almost invariably present in large numbers in all cases of carcinoma with pronounced lactic-acid fermentation.

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[1] *Arch. f. Verdauungskrankh.*, 1898, vol. lv., p. 23.

[2] *Amer. Jour. Med. Sci.*, October, 1902.

Boas reports that in some cases more or less pus is found in the stomach-contents. He found this 40 times. Personally, I have never seen such cases, and wherever I have found pus in the stomach, it was derived from some other part of the body and was present in the stomach by chance.

[Strauss[1] refers to the frequency with which pus is found in stomach-contents, and regards it, when other well-known causes are eliminated, as strongly indicative of cancer.—Ed.]

Absorption, as might be expected *a priori*, is more or less reduced in carcinoma of the stomach. Penzoldt, Faber, Quetsch, Häberlin, Wolff, Zweifel and A. Freilich, and others have performed a number of investigations into this question. All this work was done with potassium iodid, and in nearly all cases of carcinoma it was found that absorption was more or less retarded. Unfortunately, this method gives no exact picture of the perversion of absorption.

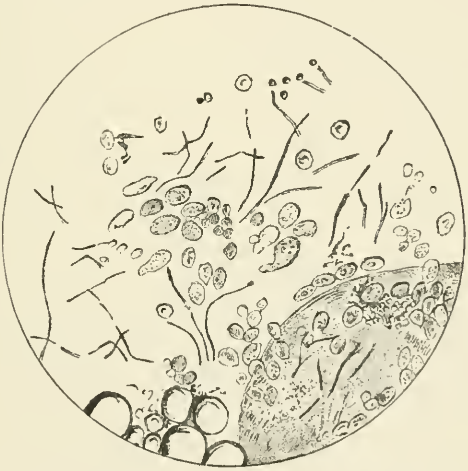

Fig. 20.—From the stomach-contents of a carcinoma case: yeast-cells and thread-like bacilli.

The urine is abnormal in different respects in cases of carcinoma. As long as the ingestion of food, the motility of the stomach, and absorption are approximately normal, the quantity excreted does not deviate much from the normal; if too little food is eaten; if there are advanced ectasy and much vomiting, however, the amount of urine excreted is naturally reduced and its specific gravity is increased. We frequently find cases of carcinoma that void only 400 to 500 c.c. in the twenty-four hours.

Another symptom that is worthy of mention has been discovered by Sticker and Hübner. These authors performed a number of investigations in my clinic and discovered that the physiologic acidity curve of the urine after eating is changed in carcinoma of the pylorus. This phenomenon is readily explained by the reduced production of hydrochloric acid.

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[1] *Berlin. klin. Wochenschr.*, 1899, No. 40.

Only within recent years have exact investigations into the excretion of urinary nitrogen in carcinoma of the stomach been published. A number of these investigations are useless, however, because the excretion of nitrogen was studied alone, and not at the same time the ingestion of nitrogen. Fr. Müller[1] and Klemperer[2] have reported some very interesting results in this direction. They found that the excretion of nitrogen is increased in carcinoma as compared with the ingestion of nitrogen; in other words, that there is increased catabolism of tissue-protoplasm. We see, therefore, that the organism loses albumin; that the protoplasm of the different organs is disassimilated. There are, of course, cases of carcinoma, or, better, patients in certain stages of carcinoma, who maintain nitrogen equilibrium for a time. This is shown by the observation that the general health and the general nutrition in many cases of carcinoma remain good for a certain period of time, and that there is not only no loss of weight, but occasionally an increase.

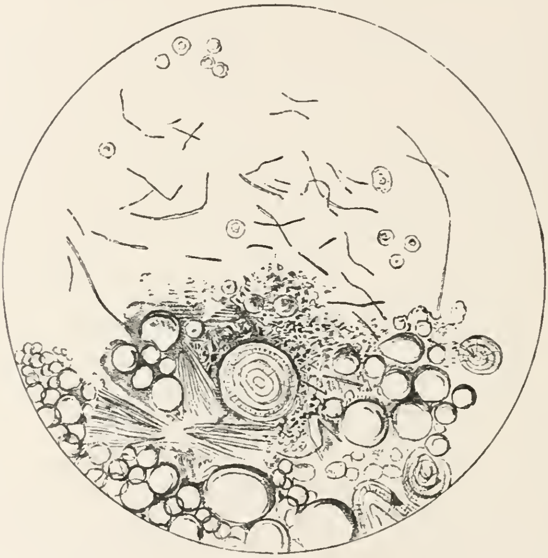

Fig. 21.—From the stomach-contents of a carcinoma case. Fat-droplets, starch-granules, blood, thread-like bacilli.

The chlorids of the urine are, as a rule, diminished in carcinoma. This is natural when we consider that the chief source of the urinary chlorids is the food. Sticker and Hübner,[3] and after them Gluzinski,[4] have shown that this decreased excretion of chlorids is particularly conspicuous in those cases where absorption is bad and the secretion of hydrochloric acid is increased. Gluzinski, therefore, argues that the determination of the absolute values of chlorin in the urine may aid us in the differential diagnosis of the question whether ectasy is caused by a carcinoma or by an ulcer scar at the pylorus. This assumption, however, has not been verified, for in every case of advanced stenosis of the pylorus the absorption of food, and consequently the excretion of chlorids, is impaired. Stroh[1] has performed a number of analyses in my clinic in cases of dilatation of the stomach with abundant secretion of hydrochloric acid. He found that the amount of sodium chlorid excreted in the twenty-four hours in one case was 0.2 to 1.5 gm.; in another case 0.16 to 0.18 gm.; in a case of carcinoma, 0.34 to 3.4, and in a second case of carcinoma, 0.24 to 1.7 gm. The absolute quantity of chlorin, we see, therefore, is reduced in carcinoma, but this decrease is without value in the differential diagnosis (von Noorden).

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[1] *Zeitschr. f. klin. Med.*, vol. xvi.

[2] *Berlin. klin. Wochenschr.*, 1889.

[3] *Zeitschr. f. klin. Med.*, vol. xii.

[4] *Berlin. klin. Wochenschr.*, 1887.

Albuminuria is quite frequently found in carcinoma; at all events more frequently than in ulcer, where it is only exceptionally found. F. Müller has summarized the histories of all the cases of carcinoma of different organs that occurred in Würzburg, and reports albuminuria in 35 per cent. of the cases. In Berlin F. Müller found albuminuria in about one-half of the cases, and determined that it was usually transitory in character. My own observations teach me that this albuminuria is, as a rule, slight and transitory.

Albumose, peptone, and occasionally diacetic acid and oxybutyric acid are also sometimes found. According to the investigations of Leo, Stadelmann, and Hoffmann, the amount of pepsin excreted in the urine is very much reduced in carcinoma of the stomach. This matter, like the question in regard to the secretion of all the other ferments, however, calls for more careful investigation.

The excretion of indican in large quantities is more important. According to Senator, this is one of the most constant symptoms of carcinoma of the stomach. Häberlin, however, did not see such uniform results in this respect as Senator. Generally speaking, the appearance of large quantities of indican in the urine may be regarded as a sign of increased proteid catabolism.

Rosenbach's reaction—*i. e.*, the appearance of a Burgundy-red color on addition of nitric acid to boiling urine—is without significance in the diagnosis of carcinoma. According to Rosenbach, this reaction is positive in serious intestinal troubles of different kinds in which absorption is reduced. It is also seen in the later stages of carcinoma of the pylorus as soon as symptoms of inanition appear. This reaction appears approximately under the same conditions as the indican reaction.

The Blood.—We have already spoken of the effect of carcinoma of the stomach on the general nutrition and the appearance of the patient. It was to be expected *a priori* that the examination of the blood would reveal certain changes. Some authors report a considerable decrease in the number of red blood-corpuscles and an increase of the leukocytes. Müller found the red blood-corpuscles reduced to 1,300,000. The proportion of white to red blood-corpuscles may increase to 1 : 90 or even 1 : 60. According to Müller, an exception to this rule is seen in those cases in which the absorption of water is interfered with; particularly, therefore, in cases of advanced stenosis of the esophagus or the pylorus. As a result of the deficient absorption of water, the blood is thickened and the number of blood-corpuscles may be increased to 5,000,000. Occasionally the red blood-corpuscles are changed and appear in the field as poikilocytes and microcytes.

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[1] *Inaug. Diss.*, Giessen, 1888.

The percentage of hemoglobin is also frequently reduced. Häberlin found it reduced one-half in the majority of cases. The specific gravity of the blood has also been found lower than normal (Peiper, Boas).

Another important symptom that Schneyer[1] discovered is the absence of digestion leukocytosis in carcinoma. According to this author, the appearance of digestive leukocytosis militates against the diagnosis of carcinoma; but, on the other hand, the absence of this phenomenon does not indicate carcinoma.

[The more recent investigations of Stengel, Douglas,[2] Osler and McCrae,[3] all go to show that no reliance can be placed upon digestion leukocytosis as an aid to the diagnosis of cancer of the stomach. It seems that only about 54 per cent. of cases gave positive reaction in this regard. It has been shown by A. Japha[4] that in young children, whether sick or well, digestive leukocytosis is not a regular phenomenon, and in them, therefore, lacks diagnostic value.—Ed.]

Complications and Sequelæ.—In the preceding sections we have already discussed the ordinary sequelæ of carcinoma of the stomach as far as the urine and the blood are concerned. In this section we will enumerate briefly a few of the less frequent sequelæ and the more important complications of carcinoma of the stomach.

Fever.—In the great majority of cases fever is absent in carcinoma of the stomach. Febrile temperatures are very rarely observed in the course of this disease. The fever may be due to a variety of causes. Whenever we find fever in carcinoma cases, we should always think of a complication; it may, for instance, be due to some inflammatory process in the vicinity of the carcinoma, to circumscribed peritonitis, to pleuritis, etc. Sometimes the patients must be studied for several days before the cause of the fever can be discovered. Finally, there are a number of cases in which no direct cause for the fever can be discovered —in which the fever seems to be directly due to the carcinoma. Cases of this kind, I must say, are rare. Hampeln[5] and Devic and Chatin[6] have recently investigated the course of the fever that is occasionally observed in carcinoma of the stomach. They found that it is intermittent in character. Wunderlich, in his well-known hand-book, mentions this peculiar fever. Hampeln emphasizes that it is not only intermittent in character, but occasionally appears in the form of distinct fever paroxysms with a stage of cold, heat, and chill, as in malaria and septicopyemia. Hampeln's statistics demonstrate that cases of this character are rare. Probably the febrile paroxysms in these cases are due to absorption of disintegration products of the carcinoma.

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[1] See *Berichte d. 66. Versamml. deutscher Naturforscher und Aerzte in Wien*; also *Berlin. klin. Wochenschr.*, 1894, No. 41.

[2] *Brit. Med. Jour.*, March 16, 1901.

[3] *Cancer of the Stomach*, 1900, p. 119.

[4] *Jahrbuch f. Kinderheilk.*, 1900.

[5] *Zeitschr. f. klin. Med.*, 1888, vol. xiv.

[6] Devic and Chatin, *Province médicale*, 1892.

Coma.—Coma is more frequently observed in carcinoma cases that are febrile paroxysms. We are indebted chiefly to von Jaksch,[1] Senator,[2] Riess,[3] Klemperer,[4] and others for our knowledge of this peculiar symptom-complex. Formerly we knew only a diabetic coma, until von Jaksch saw the same typical symptom-complex in a case of carcinoma in which the urine contained an abundant quantity of acetone. Riess and Senator have contributed much to the casuistic of this subject.

Müller and Klemperer were the first to show that in carcinoma the disintegration of body proteid is, as a rule, increased. A similar disintegration of protoplasm is found in a variety of intoxications. We need refer only to phosphorus and arsenic poisoning and to severe infectious fevers. Carcinoma is intimately related to all these intoxications—at least, in this respect. Klemperer feels justified in drawing the conclusion from this analogy that some toxic material is present in the blood of cancer cases that causes the disassimilation of organic protoplasm. Occasionally, he believes, the same poison may produce the fatal symptom-complex of the coma.

The symptoms of carcinomatous coma are essentially the same as those of any other form of coma. The patients grow apathetic, later relapse into stupor or become somnolent; they do not react to the voice, and the breathing becomes labored, deep, and stertorous. The pulse becomes small and rapid. Finally, death occurs. Occasionally epileptiform convulsions are seen during the coma. It is interesting to note that Klemperer observed a reduction in the secretion of nitrogen in two cases of coma in which, before the advent of the coma, proteid catabolism had been increased. He also discovered oxybutyric acid in the urine.

There can be no doubt, therefore, that the coma of carcinoma resembles diabetic coma in many respects. In both conditions general metabolism is perverted. This is demonstrated by the appearance of considerable quantities of acetone, and usually of diacetic acid, in the urine, and by the reduced alkalescence of the blood. In a few instances, as we have said, oxybutyric acid was also found in the urine during terminal coma. It has not yet been decided whether this coma is due to specific poisons that are produced by the carcinoma, or whether it is merely an acid intoxication.

[This subject has recently been reviewed by Osler,[5] who reports a case of coma occurring late in the history of carcinoma of the stomach, which showed diacetic acid and acetone, although no sugar was present, but which resembled closely in other respects the coma of diabetes. A perfect explanation of the phenomenon is still awaited.—ED.]

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[1] *Verhandl. d. 2. Cong. f. innere Med.*

[2] *Zeitschr. f. klin. Med.*, vol. vii.

[3] *Ibid.*, supplement to vol. vii.

[4] *Berlin. klin. Wochenschr.*, 1889, No. 40.

[5] *Johns Hopkins Hosp. Bull.*, June, 1902.

Tetany is a very rare complication of carcinoma of the stomach. This condition had, until recently, been exclusively seen in cases of ectasy with hypersecretion, but a few cases of tetany are also on record that occurred in ectasy produced by malignant tumors of the pylorus. I have observed two cases of this kind myself. For the symptoms of tetany I refer to page 163, and to the section on Hypersecretion, page 355.

Hydrops is rare in carcinoma of the stomach, and usually occurs toward the end. It generally assumes the ordinary character of marantic hydrops—that is, it begins at the ankles and gradually extends upward. It rarely reaches advanced degrees. Occasionally ascites develops, particularly if the carcinoma extends to the peritoneum or if the portal vein is compressed—for instance, by carcinosis of the lymph-glands. Occasionally venous thrombosis occurs, leading to edema of one extremity.

Metastases may appear in a number of organs, the liver being most frequently involved. Occasionally symptoms of metastases in the liver appear before the stomach symptoms are very conspicuous. The patients complain of a feeling of pressure in the epigastrium and in the region of the liver, their appetite is poor, and they become emaciated. On examination the liver will be found to be very much enlarged and nodular, its surface covered with numerous nodules. Occasionally there is icterus. If the primary carcinoma is not located at the pylorus, but at the lesser curvature, the tumor of the stomach may evade detection by palpation because it is covered by the liver. I recently observed this in a case that seems to me sufficiently interesting to warrant recording in this place:

W. N., day-laborer, age sixty-eight, admitted to the hospital on April 26, 1894, complaining of weakness, loss of appetite, and constipation. The objective examination revealed enlargement and great hardness of the liver, particularly of the left lobe. Nothing abnormal was found in the region of the gall-bladder. On account of the extraordinary hardness of the liver, carcinoma of the organ was assumed, particularly as it appeared as though ridges could be felt on the surface of the organ. The examination of the stomach-contents after a test-breakfast and after a test-meal revealed peculiar conditions. A very short time after the test-meal or test-breakfast only traces of food-remnants could be found in the stomach. The material usually consisted of coarse, lumpy particles of food that were covered with mucus, that, as a rule, occluded the sound. On lavage, only very scanty remnants of poorly digested stomach-contents were evacuated. The quantity fluctuated between 5 and 40 c.c. Free hydrochloric acid was never present; lactic acid was occasionally found. The acidity fluctuated between 6 and 64. In this case, therefore, the contents of the stomach was evacuated with abnormal rapidity, but the particles of food were poorly macerated, mechanically, and there was evidence of serious insufficiency of the chemism of the stomach. At the same time lactic acid and mucus could be found. Oatmeal soup disappeared from the stomach in a relatively short time,—three to four hours,—and we never succeeded in getting enough material at the expiration of this time to perform quantitative determinations of lactic acid. It was also discovered that the capacity of the stomach was small. More than half a liter of flour soup could never be introduced at a time; if more was introduced, vomiting occurred at once. No tumor of the stomach could be palpated. The left lobe of the liver nearly filled the whole left hypochondriac region (compare Fig. 22). Inflation of the stomach gave no positive result.

In the course of the disease the tumor of the liver increased in size (compare Fig. 22, *a*, *b*, *c*). Distinct irregularities of its surface could be felt only toward the end. Nothing abnormal was ever felt in the region of the gall-bladder. In the beginning of June the skin became yellow, but no urobilin nor bile-pigment was excreted in the urine. In the beginning of July bile-pigment was found in the urine, and the stools became acholic; finally sopor and coma appeared and continued until death. Sugar was never found in the urine, and albumin was found only toward the end.

The Autopsy.—Carcinoma of the liver with numerous nodules; a few metastatic cancer-nodules in the lung; the gall-bladder intact; carcinoma of the portal glands. A gland about as large as an apple compresses the head of the pancreas. The tissue of the pancreas is not destroyed in any place. Near the orifice of the common duct is a large and carcinomatous gland that compresses the common duct. This stenosis of the common duct by compression probably was the cause of the icterus that appeared in the last month of life.

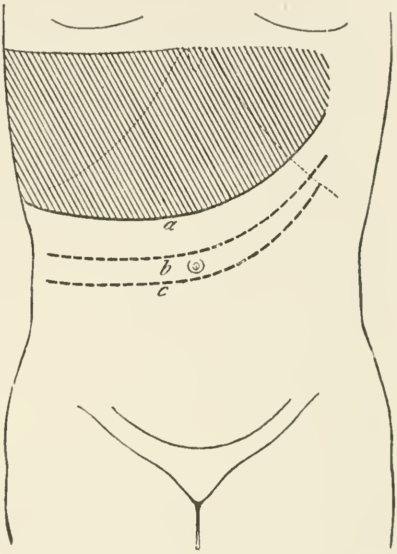

FIG. 22.—*a*, Lower boundary of the liver when the patient entered the hospital on April 26, 1894; *b*, lower boundary of the liver on June 25, 1894; *c*, lower boundary of the liver on July 8, 1894.

The changes observed in the stomach were particularly interesting. The whole organ was smaller than normal; no stenosis of its orifices could be observed. A small carcinomatous tumor about as large as an apple, and ulcerated, was found in the lesser curvature, but did not extend to the pylorus. Owing to inflammatory processes in the vicinity of the tumor retraction of the whole lesser curvature had taken place, so that in this way the size of the stomach was reduced. The musculature of the organ, at the same time, was of normal thickness, here and there hypertrophic, and this probably explains the good motility, even though there was no stenosis. The mucosa was swollen and covered with old hemorrhagic areas.

Here, therefore, the primary factor was carcinoma of the stomach; secondarily, the process extended to the liver, the lungs, and different glands. Here, too, coma carcinomatosum developed in the last days of life. Another interesting feature of this case was the fact that the carcinoma did not involve the orifices of the stomach, so that no ectasy developed.

We should make it a rule to study the secretory and motor powers of the stomach in every case of carcinoma of the liver, even though no symptoms of primary carcinoma of the stomach are present. Carcinoma of the liver, with the exception of carcinoma of the gall-bladder, is, as a rule, secondary, and is most frequently due to a primary carcinoma of the stomach. In carcinoma of the gall-bladder we can usually feel a hard, nodular, frequently painful tumor, in the region of the gall-bladder, that protrudes considerably below the margin of the liver.

Diseases of the pleura and the lungs are rare complications of carcinoma of the stomach. The left pleura is more frequently involved than the right. The amount of exudate is rarely large. Secondary carcinosis of the lung is rare, and, as a rule, produces no particular symptoms because the different nodules are too small. This was seen, for instance, in the case we have described. The mediastinal glands may also be secondarily infected, but they rarely grow so large, as in the case reported by Mathieu, that they cause severe dyspneic and asthmatic attacks.

Another rare complication is **multiple neuritis.** Klippel[1] reported 5 cases of carcinoma with multiple neuritis, 3 of which were examined anatomically. In 2 instances there was degeneration of the peripheral nerves with changes in the musculature. In 1 instance the musculature alone was changed. Auche[2] performed an anatomic examination of the nerves in 10 cases of carcinoma that developed certain symptoms of peripheral neuritis during life. In the majority of these cases the large nerve-trunks were found intact, whereas the peripheral branches were involved.

Miura[3] has recently reported a typical case of carcinomatous neuritis from Gerhardt's clinic. The patient was a man of thirty-two years, who developed multiple peripheral neuritis in the course of carcinoma of the stomach. In the beginning this complication progressed slowly, later very rapidly, and ultimately involved both the cranial and the spinal nerves. The clinical diagnosis was corroborated by a postmortem examination that revealed multiple degenerative neuritis. The central nervous system was intact. Miura assumes that in carcinoma a poison is developed, and that this may produce degenerative neuritis in persons who are predisposed. If this theory is correct, we must assume that the predisposition to neuritis is rare, as this complication is seen in very few cases.

Metastases in the eye have also been observed. Gayet,[4] for instance, reports a case of primary carcinoma of the stomach with multiple nodules in the liver, the right lung, and the choroid of the right side. Other investigators have recórded sudden visual disturbances in the course of carcinoma. Deutschmann[5] reports a case of carcinoma in which visual disturbances appeared suddenly and led to complete blindness within a few days. On autopsy a carcinoma was found in the region of the lesser curvature that was about as large as a hen's egg and was ulcerated. The ophthalmoscopic examination was negative. Deutschmann believes this amaurosis to be the result of an autointoxication from the carcinoma of the stomach.

The following case that I observed is unique. Here the carcinoma led to hour-glass contraction of the stomach.

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[1] Klippel, "Des amyotrophies dans les maladies générales chronique et de leurs rélations avec les lésions des nerfs périphériques," *Thèse de Paris*, 1889.

[2] *Revue de méd.*, 1890, vol. x.

[3] *Berlin. klin. Wochenschr.*, 1891, No. 37.

[4] *Arch. d'ophthalmologie*, vol. ix.

[5] *Beiträge zur Augenheilkunde*, published by Deutschmann, vol. i., p. 34.

Fr. Sch., agriculturist, age seventy-two, admitted to the hospital on December 29, 1893, complaining of weakness, acid belching after eating, poor appetite, and sluggishness of the bowels. Within the first few days an elongated tumor could be felt in the epigastric region above the umbilicus. The outlines of this tumor could not be distinctly determined. It did not move on respiration, and did not belong to the liver, for the free margin of the latter organ could be distinctly palpated. From its position and outline we assumed that this tumor probably belonged to the stomach. Inflation could not be performed in this case because the patient succumbed to a hemorrhage that occurred suddenly the second time the stomach was aspirated.

The probable diagnosis "carcinoma ventriculi" was made from the results of the analysis of stomach-contents performed four hours after a test-meal. Two hundred and fifty c.c. of poorly digested food were found, free hydrochloric acid was absent, and there was a distinct lactic acid reaction. Another factor that spoke in favor of this diagnosis was the sudden appearance of symptoms of peritonitis two days before death. On autopsy it was found that the tumor of the stomach could not have been palpated during life, as the upper portion of the stomach was adherent to the diaphragm. The tumor that we felt during life was a metastasis in the radix mesenterii about as large as a small adult fist. The stomach was divided into two portions by a vertical fold (hour-glass contraction), so that it presented a peculiar appearance (see Fig. 23).

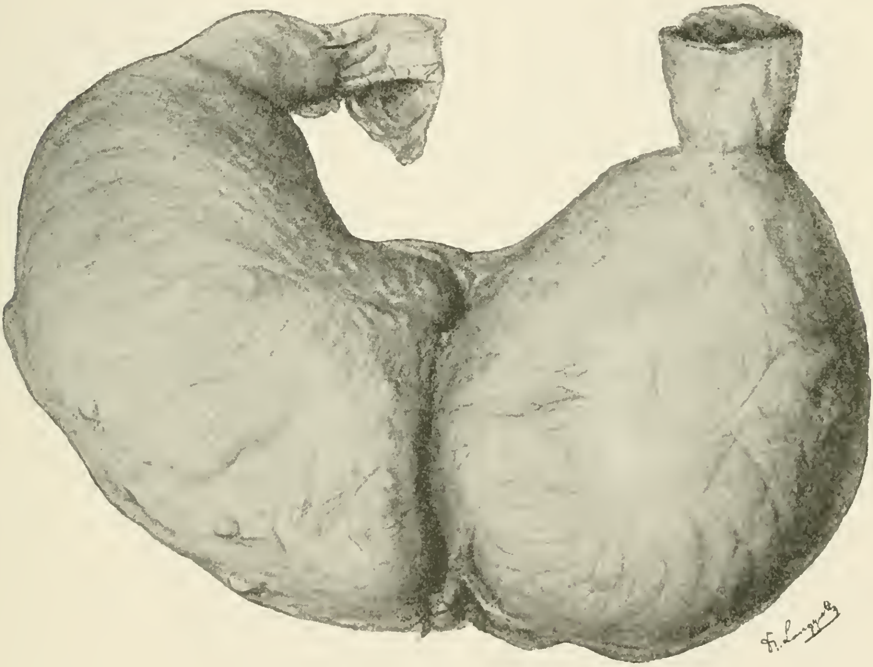

FIG. 23.

When the stomach was opened, a large tumor was found that divided the organ into two halves (see Fig. 24). Metastasis of the carcinoma, which was found to be a scirrhus, were found in the mesentery, the peritoneum, the retroperitoneal lymph-glands, and the kidneys (Fig. 23).

The most remarkable feature of this case is that the carcinoma, which started from the lesser curvature, divided the whole cavity of the stomach into two halves and in this way produced an hour-glass contraction of the organ.

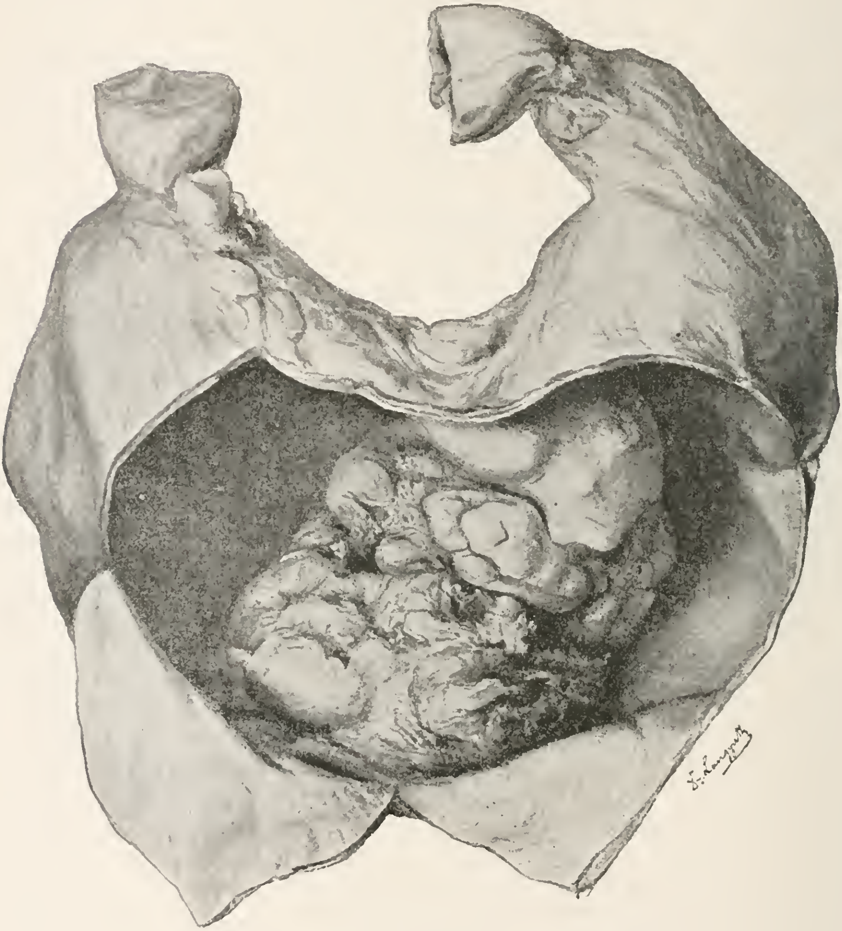

FIG. 24.

Course and Termination of Carcinoma of the Stomach.—In the section on the Symptomatology we have repeatedly described the course of this disease. In general, carcinoma of the stomach is a disease that steadily progresses from bad to worse and ultimately leads to death. For a time the process may be arrested, or the patient may even seem to be improved. Cases are quite frequently encountered in which the patient feels better for weeks or even months, in which the appetite, the general strength, and the weight not only do not decrease, but even increase. Improvement of this kind is, of course, found only in the early stages of carcinoma. Unless a clinician is experienced, he may be misled by this improvement and doubt the diagnosis of carcinoma, particularly if no tumor can be felt. If we succeed in raising the appetite, in stopping vomiting, the patient can take an appropriate and a sufficient amount of food, so that the general nutrition may improve. This occurs particularly in those cases that are not complicated by ectasy, that possess good motor powers, or, as in many cases, increased motor powers. If the diet is selected carefully in these cases, an improvement, as I have said, may be brought about. Unfortunately, it does not continue for long. Sooner or later, as the carcinoma develops, the disagreeable symptoms return, the appetite is lost, the patient does not take sufficient nourishment, and soon loses strength. If the carcinoma is situated at the pylorus, the symptoms of stenosis rapidly develop, and gradually more and more food is retained and less of the ingesta assimilated.

In some instances transitory improvement may be brought about by a disintegration of the neoplasm. This usually relieves the stenosis of the orifices of the stomach and renders them more patent. This improvement is seen particularly in carcinoma of the cardia. Here we quite frequently see disintegration of the neoplasm late in the disease; when this occurs the passage becomes free and the patients may even be able to swallow solid food. The same may occur in carcinoma of the pylorus; here, too, disintegration of the neoplasm causes disappearance of the symptoms of stenosis and allows the food to pass into the intestine without difficulty. As a rule, however, all this occurs in late stages of the disease and at a time when the loss of strength is already very considerable.

As the carcinoma develops the patients continue to lose strength and weight, the adipose layer gradually disappears, the musculature grows flaccid and atrophic, the appetite is finally lost altogether, so that the patients can only with difficulty be persuaded to eat anything. In the majority of cases there is a particular aversion to meat.

The course of the disease naturally varies in different cases. Much will depend on the location and the type of the carcinoma and on the presence or absence of ectasy or of other complications. It is impossible to describe all the different pictures that carcinoma may produce. No two cases resemble each other in all details, although, of course, the main symptoms are the same in all. In cases of carcinoma of the cardia, symptoms of stenosis and vomiting, or rather of regurgitation of food, are, of course, most conspicuous. In carcinoma of the pylorus leading to stenosis and ectasy obstinate vomiting may be the most prominent symptom unless the stomach is relieved by methodic lavage. In other cases, again, progressive anorexia and progressive loss of strength are the chief symptoms. The decline of the patient continues until, finally, inanition terminates the scene. In other instances one of the complications that we have described or some intercurrent disease may lead to death.

A rare termination of carcinoma of the stomach that I might mention is perforation into the abdominal cavity. In other cases, again, the carcinoma involves neighboring organs and leads to perforation of the pleura or of the pericardium. In still other cases abnormal communications are formed between the stomach and other hollow organs—for instance, the intestine. In very rare instances perforation of the carcinoma of the stomach through the abdominal wall has been known to occur. Brinton found only one perforation of this kind in 507 cases of carcinoma of the stomach. Mislowitzer has recently collected 17 cases of this kind, one of which he observed himself. In all these cases the perforation was preceded by a softening of the tumor, by fluctuation, and by inflammatory reddening of the cutaneous covering. Perforation occurred either spontaneously or by incision into the softened area. In the majority of cases there was only one fistula; in three cases, however, several fistulæ were found. In many of the cases viscid pus poured out of the fistula, and in 8 cases food-particles could be found in the canal. The patients survived the perforation for three days to three months. The carcinoma in these cases was usually of the medullary type.

Wunschhein[1] has reported a case that is unique. In his case the stomach was torn at the lesser curvature by the perforation of an esophageal, or, better, cardial, carcinoma into the aorta. This led to a sudden distention of the stomach with blood and rupture of the organ.

Achard[2] reports a case of carcinoma of the pylorus and of the lesser curvature that was characterized by a number of extraordinary symptoms—namely, an intraperitoneal phlegmon that led to an umbilical fistula and, by purulent degeneration, to secondary cancer nodules in the liver. Death in this patient resulted from purulent peritonitis.

The duration of the disease varies greatly. In the majority of cases it is impossible to determine how long the disease lasts, chiefly because the time of onset cannot be determined with accuracy. The average duration is calculated as from one to two years; hard carcinomata, as a rule, pursue a longer course than soft carcinomata, as the latter show a greater tendency to degeneration and ulceration. Undoubtedly many cases survive longer than three years, whereas in others, again, the course of the disease is much shorter. It is established nowadays that operative interference at the right time may prolong life considerably even if it is impossible to remove the cancer. Cases of this kind, however, must be considered rare for the present.

Diagnosis.—The diagnosis of carcinoma of the stomach is undoubtedly more certain nowadays than it was twenty years ago. If we compare the diagnostic adjuvants at our disposal at that time with those that we possess to-day, this will not surprise us. I need only refer to the significance of the chemical analysis of the stomach-contents, the determination of the motor powers of the organ, and artificial inflation, to emphasize this point, for these measures enable us nowadays to determine that a tumor belongs to the stomach with much greater certainty than any other methods that are in vogue. Notwithstanding all these diagnostic aids we quite frequently encounter cases in which the diagnosis is difficult, even impossible. We must never forget that there is no single symptom that is absolutely characteristic for carcinoma unless we wish to consider the appearance of characteristic tumor-particles in the openings of the sound or in the vomit such a criterion. Such particles, however, are found so rarely that we need hardly consider this contingency. I will not speak of gastroscopy in this place, because this method of examination is applicable only to very isolated cases.

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[1] *Prag. med. Wochenschr.*, 1893.

[2] *Méd. moderne*, 1894, No. 79.

We should make it a rule never to render a diagnosis from one examination alone. The only way to avoid error is to examine the case repeatedly. This applies particularly to the chemical analysis of the stomach-contents. A single examination may frequently yield misleading results. The absence of free hydrochloric acid, above all, should never lead us to assume that the production of gastric juice is reduced or stopped, particularly in those cases in which advanced ectasy exists. If, on the other hand, free hydrochloric acid can be found even once, this finding possesses considerable significance. At the same time, as we have already shown, the presence of free hydrochloric acid does not necessarily exclude carcinoma.

The diagnosis of carcinoma, then, should never be made from one single symptom, for there is no single pathognomonic symptom of this disease. The presence of abundant quantities of lactic acid is as little pathognomonic as the absence of free hydrochloric acid. We can make a reliable diagnosis only if several symptoms are present. The more symptoms of carcinoma we discover, the more certain will be the diagnosis. It is an easy matter, for instance, to arrive at the diagnosis carcinoma of the stomach if the patient is old and cachectic, if there is advanced ectasy, if the stomach-contents presents a coffee-ground appearance, if free hydrochloric acid is absent, if there is profuse lactic-acid fermentation, and if we feel a tumor in the region of the pylorus. Even if any one of these symptoms is absent, however, the diagnosis is rendered difficult. Let us assume, for instance, that we have a tumor, cachexia, emaciation, ectasy, hematemesis, and that the analysis of the stomach-contents reveals the presence of abundant quantities of free hydrochloric acid and the absence of lactic acid ; in a case of this kind the diagnosis may be doubtful, for we may be dealing with cicatricial stenosis of the pylorus from an ulcer that has led to the development of a palpable tumor and to secondary ectasy. In addition there may be a recent ulcer in this case ; or, again, we may be dealing with hypersecretion combined with ulcer and ectasy and muscular hypertrophy at the pylorus ; or, finally, we may be dealing with an incipient carcinoma that has developed on the basis of a cicatrized ulcer, producing stenosis of the pylorus. What are we to decide in this case ? On the one hand, we must consider the appearance and the general consistency of the stomach-contents; on the other hand, we must study carefully the history and the course of the disease. If the case is one of hypersecretion, we will find abundant quantities of gastric secretion in the stomach even after fasting; and if it is a carcinoma that is developing on the basis of an ulcer, we will learn from the history that the disease has existed for a long time, and that symptoms of ulcer were formerly present. The only way in which to arrive at a definite decision in cases of this kind is to repeat the examination at different times of the day and to study the case carefully for a long time.

Let us assume the following case, which is simpler, and which we are apt to encounter more frequently in practice: The patient may be a man of forty or thereabouts, with a nervous disposition, who gives us the following history. For three months or thereabouts he has been complaining of loss of appetite, frequent belching, a bad taste in the mouth, general lassitude, and lack of mental energy. He has not noticed that he is losing flesh. We examine the patient and find nothing abnormal. The patient is not particularly strong, and possibly looks a little pale, but is not cachectic. The stomach is enlarged, but no tumor can be discovered. The whole region of the stomach is slightly sensitive to pressure, but there is no circumscribed painful pressure-point. We administer a test-meal and aspirate the stomach-contents five and a half hours later, and find the stomach empty. We administer a second test-meal and aspirate the stomach-contents three hours and a half later. Again we find a very slight residue, consisting largely of coarse particles of food that are covered with mucus, and, in addition, a number of coarse, only slightly digested morsels of meat. Chemical examination reveals the complete absence of free hydrochloric acid, complete loss of the peptic powers of the gastric juice, and a very weak lactic-acid reaction or none at all. What are we to diagnose? All that we can say is that we are dealing with some disease of the stomach that does not involve the motor powers of the stomach, but produces serious disturbances of the peptic powers of the organ. We may think of nervous dyspepsia, of subacute severe gastritis, of achylia gastrica, or of incipient carcinoma, but how are we to make a differential diagnosis between all these possible conditions? The only way in which to arrive at a positive decision is to keep the patient under observation for a long time and to perform examinations of the stomach-contents repeatedly.

The least probable diagnosis of all is nervous dyspepsia. If, on subsequent examination, we find that free hydrochloric acid remains absent from the stomach-contents, and that the peptic power remains reduced, also that the secretion of mucus continues, the diagnosis nervous dyspepsia will become still more improbable.

The differential diagnosis between simple atrophic gastritis and incipient carcinoma is still more difficult. The most important feature here is the duration of the disease. Carcinoma may lead to a great reduction of the gastric secretion in a short time, whereas chronic gastritis requires a much longer time to produce a permanent reduction in the secretion of gastric juice. Occasionally, however, we encounter cases of primary atrophy of the gastric mucosa that remain latent for a long time and produce no serious distress. As long as the motor powers of the stomach are intact in these cases, and as long as there are no intestinal disturbances, the general nutrition is not affected by this atrophy of the mucosa, even though the secretion of gastric juice is greatly reduced. This is due to the fact that the intestine vicariously assumes the rôle of the stomach. In carcinoma, on the other hand, we usually find that the general nutrition is impaired much earlier in the disease, even in those cases in which the motor powers of the organ remain relatively good.

We see, therefore, that it is frequently impossible to make a differential diagnosis between genuine atrophic gastritis and carcinoma unless we observe the patient for a long time. If it is found that the peptic powers continue to decrease, and if, in the subsequent course of the disease, lactic acid is found; if, furthermore, numerous long bacilli and slight admixtures of blood are found several times in the stomach-contents; and if the patient, finally, emaciates rapidly, the diagnosis carcinoma becomes more and more probable. If at the end of a certain time a tumor can be palpated, the diagnosis becomes positive; but even if no tumor can be felt, the diagnosis of carcinoma becomes more and more probable if the patient continues to emaciate, even though the motor power of the stomach remains good. Primary atrophy of the gastric mucosa, as we have said, may persist for years without impairing the general health of the patient; only if atony occurs or if intestinal disturbances develop in the course of the disease does the general health of the patient become very much impaired.

It may be considered established that free hydrochloric acid may also be absent in acute and subacute gastritis; but if gastritis of this character is of recent date, we will rarely find that the secretion of gastric juice remains greatly reduced for a prolonged period of time. In these cases the absence of free hydrochloric acid as revealed by one analysis of the stomach-contents does not enable us to render a decision. The important criteria in this case are the constancy of the symptoms, the duration of the disease, and the rapidity of its development.

These few examples may teach us how difficult it is to make a diagnosis, and show us, at the same time, that one symptom may acquire a different significance according to the general group of symptoms it is combined with. It also shows that the duration of the disease and other factors must be considered. Only if we study all these points and the connection of symptoms shall we be able to render an early diagnosis; it is particularly important that we should do this, for the only chance of benefiting these patients is to render a diagnosis as early as possible. If all the important symptoms, as tumor, advanced cachexia, abundant quantities of lactic acid, absence of free hydrochloric acid, coffee-ground vomit, etc., are present, the diagnosis is easy, but it has about the same significance, as far as the patient is concerned, as a postmortem diagnosis.

Errors in diagnosis may be made in two directions. On the one hand, carcinoma may frequently be overlooked; on the other hand, chronic or subacute gastritis, anemia, nervous dyspepsia, even hypersecretion and still other pathologic conditions of the stomach, may be mistaken for carcinoma. The only way in which to avoid such errors is to perform all the methods of examination that we have described. We should never attach too much importance to one single symptom, but always consider the whole syndrome. It would lead us too far were we to enter into a discussion of single symptoms and their particular significance in this place; we have already done that in previous paragraphs. I do not believe that the diagrammatic tables of diagnosis that we find in so many text-books are of much value; nor do I think that we can make a diagnosis by placing diagrammatically symptoms of ulcer, gastritis, and nervous dyspepsia in juxtaposition to those of carcinoma. Errors of diagnosis are not, as a rule, made from lack of knowledge of these more important symptoms of the different diseases, but from wrong interpretation of the examinations that are made. Of what use are these diagrammatic schemes if a tumor is thought to be of the stomach when it arises in some other organ; or if ectasy is diagnosed because the greater curvature happens to extend below the umbilical line, when subsequent examination of the stomach reveals that the organ is not enlarged at all, but only displaced downward, so that this ectasy was a simple gastroptosis? It is particularly in this direction that most diagnostic errors are committed. I think every practitioner who has had much experience will indorse this statement. Of what use are such statements as "carcinoma is usually a disease of advanced years; ulcer usually appears in young subjects; nervous dyspepsia and gastritis occur at any age"—when we remember that carcinoma occurs in children and young people, and that ulcers are frequently encountered in old persons?

From a statistical point of view, the fact that this or that disease occurs with greater frequency at this or that age is interesting; practically, however, this knowledge helps us little in individual cases, particularly as we know that the disease may occur at any age. The same applies to statistics that teach us that in certain diseases a certain symptom is rare, and that the same symptom is frequent in another disease. As all these points are of no practical importance whatever, I shall not waste time in formulating diagrammatic schemes of diagnosis, however fascinating they may be to the beginner. All the really essential points I have discussed thoroughly in the section on Symptomatology.

It seems hardly necessary to describe the points in the differential diagnosis between ulcer and carcinoma. The symptoms are so pronounced in either disease-form that the two conditions can hardly be confounded; this could occur only if the symptoms are not typical. The diagnosis can never be made by studying one of the schemes mentioned above, and seeing whether more symptoms correspond to ulcer than to carcinoma. Beginners are in the habit of making a diagnosis in this way; but this is altogether wrong, for even a single symptom that does not fit into the frame of the whole symptom-complex of the disease that seems to be present should lead us to doubt the diagnosis. We should not, therefore, ask whether more ulcer symptoms or more ordinary cancer symptoms are present in any given case, but whether all the symptoms fit into the frame of the disease we assume to be present; cachexia, for instance, hematemesis, vomiting, emaciation, may all be absent and still the diagnosis carcinoma be correct, because the whole symptom-complex, the development of the disease, its duration, and its course can be interpreted to signify only carcinoma.

Every diagnosis should be based on a careful examination. Before rendering a definite diagnosis, it should not only always be determined whether a tumor is present, but also whether it belongs to the stomach. Ectasy and gastroptosis should not be confounded with cancer of the stomach, because we have definite methods of differentiating these conditions. But even if we perform all the examinations outlined, and are thorough masters of the technic of these examinations, it may happen that it is impossible to make a diagnosis unless the patient is observed for a long time. This applies particularly to the differential diagnosis between gastritis and cancer. Then, there will always be cases in which we make mistakes. If the diagnostician is conservative and careful, he will, of course, prefer to make no diagnosis at all in doubtful cases.

Guinard collected 113 cases of carcinoma of the stomach from the *Bulletins de la Société anatomique de Paris*, only 34 of which were diagnosed. I consider this an exceptionally small proportion, and believe that unnecessary errors in diagnosis must have been made in this series. No one, of course, will deny that we occasionally encounter cases of latent carcinoma. This is natural, for where a disease of the stomach produces no symptoms, we can, of course, make no diagnosis.

Bouveret reports the case of a woman of twenty-nine years, who entered the hospital with bronchopneumonia and died there of this disease. Before the onset of the pneumonic symptoms she had never suffered from any gastric disturbances, nor was she emaciated. On autopsy a scirrhus of the anterior wall of the stomach, as large as half an adult hand, was found.

Latent cases of carcinoma like this simply cannot be diagnosed. In very old men in particular carcinoma of the stomach seems to run an altogether latent course, especially if neither of the orifices of the stomach is involved in the carcinomatous process.

There is another class of cancer cases in which the symptoms that are ordinarily considered characteristic for carcinoma are absent, but in which dyspeptic disturbances, loss of appetite, belching, and general weakness appear. I do not think that these cases belong properly to the group of latent carcinomata. If the patients are advanced in years; if no trauma or other injury has preceded the onset of the dyspeptic symptoms; if the symptoms have developed slowly and insidiously, we should always consider the possibility of cancer, even though all the symptoms are very mild. The stomach-contents should, at all events, be repeatedly analyzed in all such cases.

Although carcinoma is rarely encountered in young persons, youth by no means excludes carcinoma; for this reason the age of the patient is no diagnostic criterion in any given case. According to Mathieu, carcinoma is found in persons under thirty in 1 per cent. of all cases. In young subjects the course of carcinoma in general is more rapid, and deviates in other respects from the course of the disease in older people. In young subjects, for instance, anorexia and cachexia may remain absent for a long time. The patients look pale and anemic, but do not present the yellow, decayed appearance that we are accustomed to see in carcinoma; ascites and anasarca, according to Mathieu, are more frequently found in young persons than in old subjects. This is due to the fact that in the former patients peritonitis or general peritoneal carcinosis usually complicates the disease.

There are certain rare cases that merit particular mention in which carcinoma presents the picture of a severe anemia. Carcinoma *per se*, as a rule, leads to anemia sooner or later; there are, however, certain cases of carcinoma in which the anemic symptoms are conspicuous from the beginning and seem to be altogether out of proportion to the mild dyspeptic symptoms that are complained of at the same time. This disproportion frequently leads to the diagnosis of severe anemia. The diagnosis of carcinoma in these cases is frequently missed, particularly where the more important symptoms of carcinoma, as hematemesis, ectasy, and tumor, are absent. The diagnosis may be very difficult, particularly if the patients are young. In anemia, it is true, the adipose layer remains fairly well preserved, even though the patients look very pale, whereas in carcinoma the panniculus adiposus usually disappears early in the course of the disease. In simple anemia, moreover, we frequently hear the well-known murmurs over the heart and the veins. In carcinoma these abnormal heart- and vessel-sounds are frequently absent. The most important information, however, is given us by an examination of the gastric juice, and the results of this analysis alone frequently enable us to make the differential diagnosis. To judge from my personal experience, free hydrochloric acid is not, as a rule, absent in anemia; on the contrary, we frequently find hyperacidity. It would be interesting to examine the digestive powers of the stomach in every case of severe anemia. In this particular instance a study of the gastric chemism has additional diagnostic importance. If it can be demonstrated that the secretion of gastric juice is normal, or that there is hyperacidity, the diagnosis of carcinoma will be rendered very doubtful. Inversely, of course, the secretion of gastric juice may be very much reduced in simple anemia; in other words, there may be achylia gastrica due to primary atrophy of the gastric mucosa.

Cases that are complicated with ascites also present diagnostic difficulties. This complication is seen particularly if there is carcinosis of the peritoneum, if the portal vein is compressed, and if similar conditions obtain that lead to the accumulation of fluid in the peritoneal cavity. If at the same time the inguinal glands are indurated; if the patients are old subjects with cachexia, we are justified in assuming that a carcinomatous process is going on. Similar conditions, it is true, are seen in tuberculosis of the peritoneum. If the ascitic fluid is hemorrhagic, this may be considered another factor in favor of carcinoma; but even if we succeed in demonstrating the presence of numerous tumors in the abdomen after withdrawal of the ascitic fluid, and if we can demonstrate that they are carcinomatous, we are not justified in diagnosing the presence of a carcinoma of the stomach even though dyspeptic symptoms are present. If there is multiple carcinomatosis of the peritoneum, it is frequently altogether impossible to determine the form and position of the stomach. Here, too, analysis of the stomach-contents is essential to the diagnosis.

The differential diagnosis between carcinoma and tuberculosis of the peritoneum is, as a rule, not difficult. The latter disease is more frequent in youth than in old age, its course is slower, and the process may remain stationary for prolonged periods of time. In the majority of cases, particularly if the stomach-contents is analyzed, it will be found an easy matter to differentiate tuberculosis from carcinoma.

Hübner[1] reported the following case some years ago from my clinic, and I quote his report in this place in order to show the difficulties that may be present if ascites complicates carcinoma:

The patient was a woman of fifty-five years. The first symptom that she complained of was swelling of the abdomen. This condition soon necessitated paracentesis. She stated that the fluid aspirated was hemorrhagic. Soon after the first puncture the abdomen became enlarged again, the appetite became impaired, and the patient began to emaciate. She never complained of pain.

Examination when she entered the clinic revealed the following: Cachectic subject, with icterus, ascites, and edema of the abdominal walls. No abnormalities in the liver nor the spleen. While she was in the hospital she repeatedly vomited coffee-ground material. The appetite was soon completely lost, and vomiting occurred after each meal. She finally died of general inanition. The stomach-contents was repeatedly examined, and free hydrochloric acid found on each occasion.

The following diagnosis was made: Carcinomatosis of the peritoneum; carcinoma of the liver; no carcinoma of the stomach. The starting-point of the carcinomatous process remained obscure. Careful palpation of the abdominal organs was impossible, owing to the ascites. Even though this patient had suffered from several attacks of hematemesis and had been afflicted with severe dyspeptic disturbances, the diagnosis carcinoma of the stomach seemed improbable, owing to the constant presence of free hydrochloric acid.

Autopsy revealed carcinoma of the gall-bladder, of the lymph-glands, of the porta hepatis, of the mesentery, and of the peritoneum. The hematemesis was produced by ruptured varices of the esophagus.

If we had aspirated the ascitic fluid in this case, we would have discovered another apparent clue for the diagnosis of carcinoma, for on autopsy we found that the pyloric portion of the stomach was adherent to the carcinomatous gall-bladder and formed a circumscribed tumor that protruded below the margin of the liver. If we had found this tumor in the region of the pylorus, we would probably have made the diagnosis "carcinoma of the stomach" on the basis of this symptom. This case teaches us, on the one hand, how difficult it is to make the diagnosis if ascites is present, and, on the other, how important it is to study the totality of the symptoms. It also shows us how important a single symptom may be. In this case, at all events, the presence of free hydrochloric acid led us to assume that the stomach was not involved in the carcinomatous process. In addition we are able to determine that no ulcer had preceded the symptoms then present. All our assumptions in this instance were corroborated by the autopsy.

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[1] *Berlin. klin. Wochenschr.*, 1886, No. 13.

If ascites is present, it is, as a rule, impossible to palpate the boundaries and the position of the stomach unless the ascitic fluid is removed by paracentesis. The same applies to the discovery of tumors of the stomach and of other abdominal organs.

The case that we have just recited also demonstrates that vomiting of coffee-ground material is no positive sign, for here the coffee-ground material did not come from the stomach, but from the ruptured varix of the esophagus; from this point the blood poured into the stomach and was finally evacuated by vomiting. This symptom, nevertheless, is valuable, and I believe we should think of carcinoma in every case in which coffee-ground material is vomited, but should, of course, refrain from making a diagnosis unless all the other symptoms indicate carcinoma. If certain symptoms are presented that do not coincide with this diagnosis, we should be conservative in rendering a decision. The hemorrhages in carcinoma are, as a rule, small; the blood comes from small arteries, and remains in the stomach for a long time, hence the peculiar coffee-ground appearance. Severe hemorrhages occur very rarely. If much blood is poured into the stomach, it is immediately evacuated, so that in these instances the blood is light red in color.

I refer to the section on the Symptomatology for discussion of the diagnostic significance of carcinomatous cachexia. The longer the carcinoma exists, the greater the impairment of the secretory, and particularly the motor, powers of the stomach, and the more pronounced the cachexia. If the orifices of the stomach are not involved by the carcinomatous process; if the motor power of the stomach remains intact for a long time, so that the small intestine can vicariously assume the rôle of the stomach, cancer patients may present a relatively healthy appearance for a long time. The sooner the motor power of the stomach becomes impaired, the more rapid the development of cachexia. Cachexia, therefore, may be considered an index of the disturbance of nutrition. However important cachexia may be, the absence of this symptom does not necessarily exclude carcinoma. In the early stages of the disease cachexia is frequently absent or very slight, and it is particularly at this time that a diagnosis should be made.

The presence of a swelling is usually considered a very important symptom.[1] On the one hand, however, tumors of other organs may seem to belong to the stomach; on the other hand, even large tumors of the stomach may not be palpable, or palpable only at certain times. In the earlier stages of carcinoma a tumor is, as a rule, absent.

I have already mentioned a case, when discussing the symptoms of carcinoma, in which the tumor could be felt only when the patient was lying on his back. I have quoted another case in which carcinoma was diagnosed a long time before the tumor could be discovered, and have, finally, mentioned a number of cases in which the tumor was altogether inaccessible to palpation, although it had attained a considerable size.

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[1] Penzoldt recommends examination under an anesthetic (chloroform) in order to render the palpation of the tumor easier.

Tumors that are situated at the lesser curvature, particularly if they are small, may easily escape detection. If the liver is situated in front of the tumor, the latter may also frequently remain hidden. Tumors of the posterior wall can usually be felt only at certain times and under certain conditions. I recently had a case of this kind under my care.

W. Gr., a country letter-carrier fifty-five years old, had always been perfectly healthy, with the exception of mild rheumatic symptoms. About half a year before he entered the clinic he complained of gastric symptoms for the first time. He was admitted on April 24, 1894. He was unable to eat different articles of food, and complained a good deal of heartburn; later even a small amount of food produced distention of the gastric region and pyrosis. He never complained of violent pain. Whenever the patient ate more than usual, vomiting occurred. As soon as the patient got rid of the stomach-contents by vomiting he felt relieved. The appetite was moderate.

Examination revealed a very anemic and a very much emaciated subject. The epigastric region was retracted. A hard, movable tumor about as large as a small adult fist was felt in the pyloric region. At times the stomach seemed very much distended. The organ occupied an almost vertical position, and was freely movable on respiration (see Fig. 25). Distinct peristaltic movements extending from left to right could frequently be seen. The lesser and the greater curvature could occasionally be seen when the stomach became distended. The greater curvature was situated about a finger's breadth below the umbilicus. The stomach throughout its whole extent had a uniformly elastic feel. The portion of the organ in which the tumor was located, of course, felt harder. A distinct succussion-sound could be produced over the whole stomach. It was noticed that the tumor became distinctly palpable as soon as the stomach became spontaneously distended or was artificially inflated. Under these conditions it was almost impossible to palpate the swelling, whereas the tumor could be very distinctly palpated when the stomach was empty.

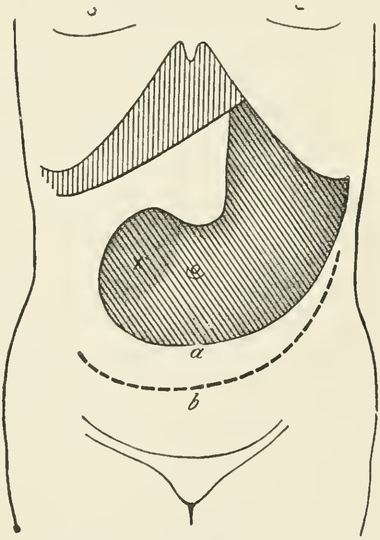

Fig. 25.—*x*, Tumor; *a*, lower boundary of the stomach; *b*, same on deep inspiration.

The great respiratory motility of the stomach, coupled with the fact that the lesser curvature was so distinctly visible, led us to suspect a vertical dislocation of the stomach. We were strengthened in this view by the observation that the tympanitic area of the stomach extended almost to the fifth rib on the left side. An analysis of the stomach-contents revealed the absence of free hydrochloric acid and the presence of much lactic acid. Even after the administration of oatmeal soup abundant quantities of lactic acid were found. Toward the end the patient repeatedly vomited coffee-ground material. Death ensued three weeks after admission.

The clinical diagnosis, carcinoma of the posterior wall of the stomach in the region of the pylorus, was made. We assumed that the tumor

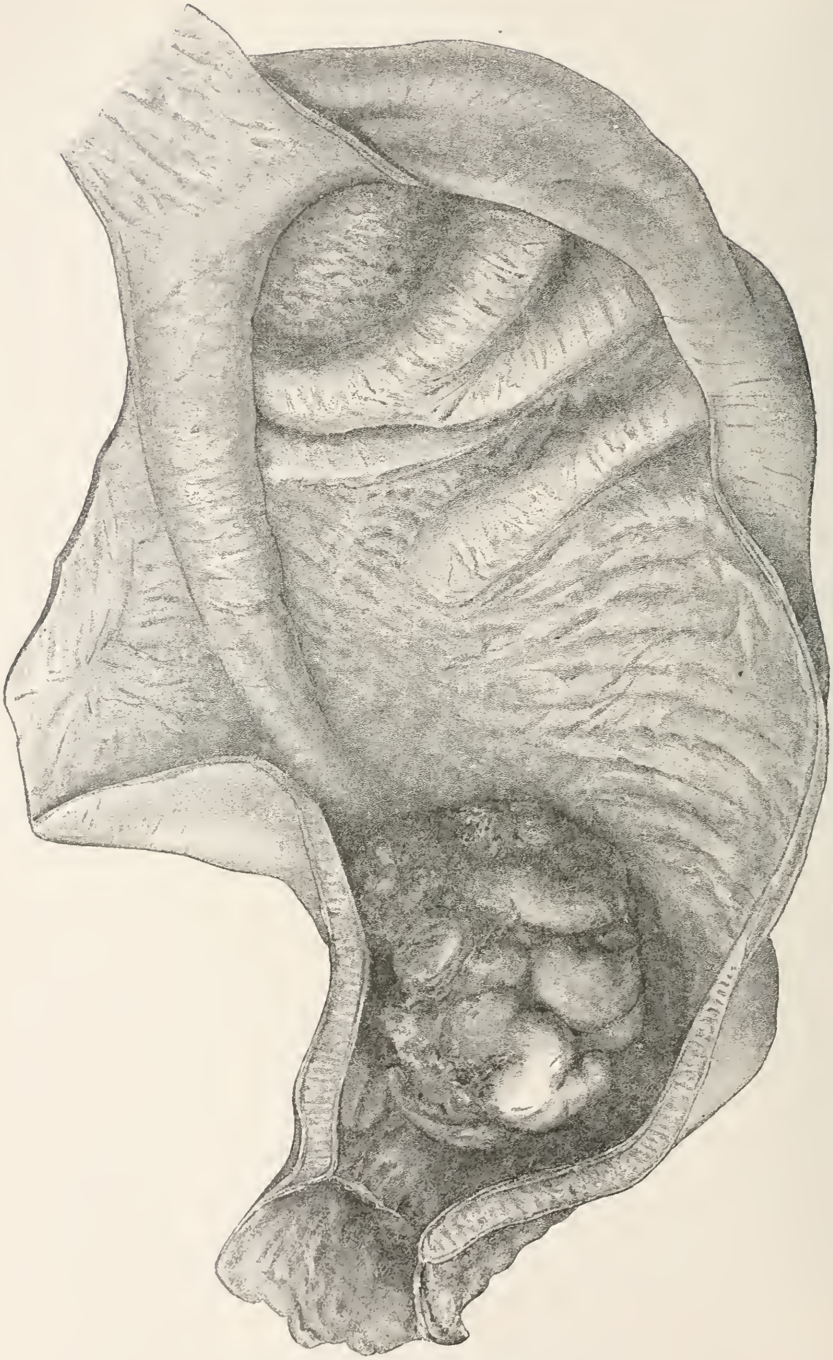

FIG. 26.—W. Gr., carcinoma of the pyloric region.

was located in this position because it disappeared as soon as the stomach became spontaneously distended or was artificially inflated, and reappeared when the stomach was not distended. At this time, in fact, it could be distinctly palpated and its outlines determined. This assumption was corroborated by the autopsy (see Fig. 26). I consider the symptom we have described—namely, the appearance of the tumor when the stomach was empty and its disappearance when the organ became distended—a very valuable point in the diagnosis of tumors of the posterior wall.

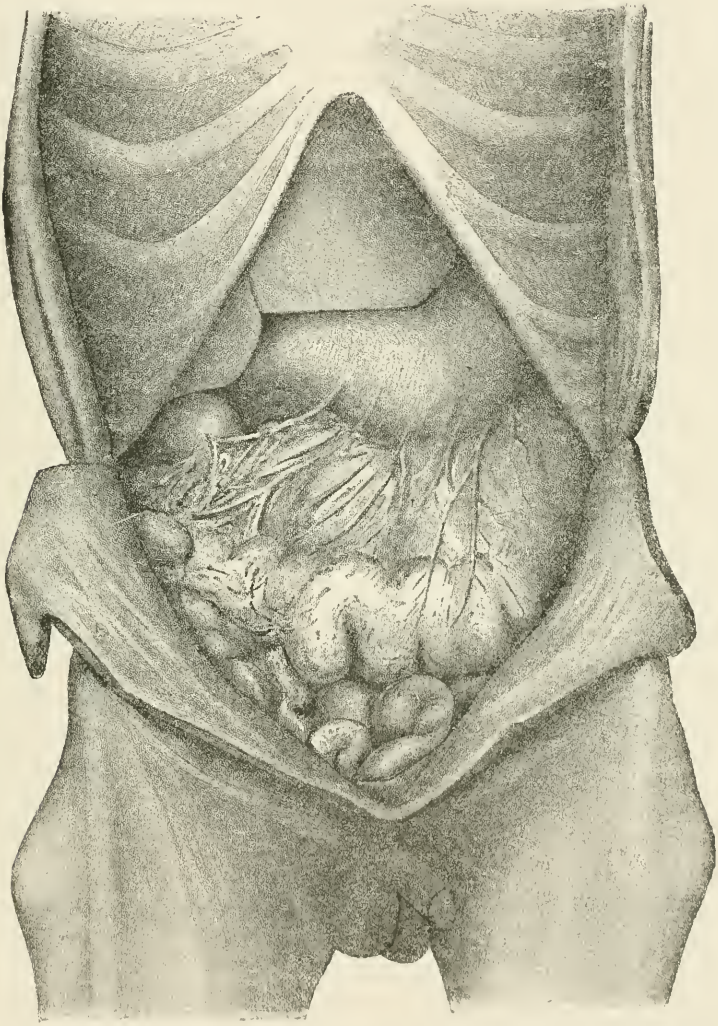

FIG. 27.—W. Gr., carcinoma of the pyloric region. Cauliflower growth of the posterior wall. Vertical position of the stomach. Ptosis of the transverse colon. Situs.

This case is interesting in another respect—namely, in regard to the great respiratory motility of the stomach. The lower boundary of the stomach, in fact the whole organ, moved downward for two or three finger's breadths when the patient inspired deeply in the dorsal position. The outlines of the stomach became clearly visible whenever the organ was spontaneously or artificially inflated. A portion of the lesser curvature could always be distinctly recognized. Compare Figs. 27 and 28, which represent the postmortem appearance. In Fig. 27 the position of the organs when the abdominal cavity was opened is depicted. In Fig. 28 the ribs are taken away and the liver is pulled back. The only explanation for the appearance of the lesser curvature was ptosis or vertical dislocation of the stomach. The only explanation of the great respiratory motility of the organ was either adhesion of the pylorus with the liver, or a close approximation of the stomach to the diaphragm. The great motility of the tumor argued against the former supposition; the visibility of the lesser curvature and the low position of the pylorus against the latter.

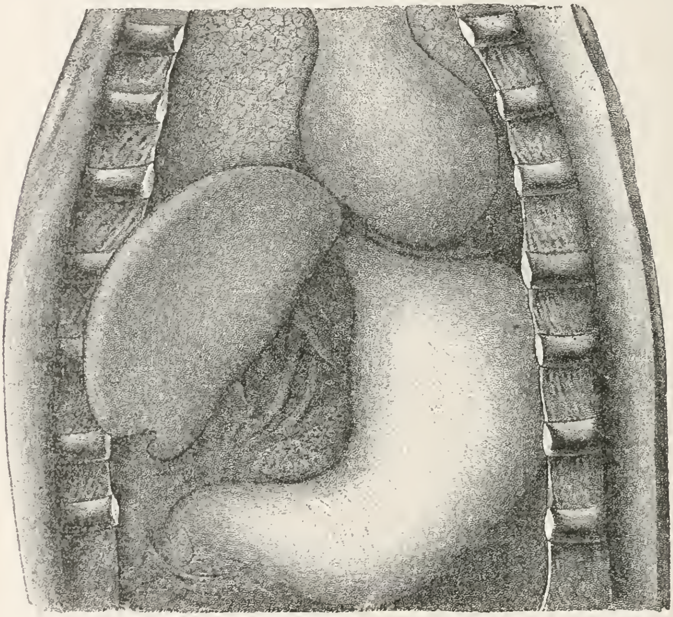

FIG. 28.—W. Gr., carcinoma of the region of the pylorus. The liver is pulled back. Vertical position of the stomach.

Within recent years numerous investigators have expressed the opinion that the stomach possesses respiratory motility only if it is in a normal position, whether it is dilated or not, but that the stomach when in an abnormal position does not possess this motility. These statements were made on the basis of transillumination experiments. I do not think that this view in so generalized a form is correct. In our case the lesser curvature was visible, and this certainly indicated that there was gastroptosis. The dislocation of the stomach was unilateral, however, as was shown by the autopsy; in other words, there was a vertical dislocation, the fundus remaining in contact with the diaphragm. We see, therefore, that the respiratory motility of the stomach need not be altered in any respect, even though the organ occupies a vertical position. It is probable that in our case the vertical dislocation of the stomach was acquired, and that it was due to the traction exercised by the tumor.

Incidentally I wish to call attention to the fact that the portion of the organ that corresponded to the hypertrophied pyloric region showed particularly active and distinct peristaltic movements. This is shown very clearly in Fig. 26.

There is active controversy in regard to the motility or non-motility of tumors of the stomach, and I have already discussed the more important aspects of this question in the section on the Symptomatology. As a rule, the respiratory movements of the diaphragm are transmitted to the stomach and to any tumors that may be present in the stomach, provided the organ is situated in its normal position and is not anchored in some place by adhesions. The motility of the stomach is, therefore, always transmitted; for the stomach merely follows the respiratory excursions of the diaphragm. The amplitude of these respiratory excursions varies according to the type of breathing, the intra-abdominal pressure, the tension of the abdominal walls, and numerous other factors. I think that the respiratory motility of the stomach, and particularly of tumors of the stomach, is not so pronounced as in tumors of the liver and of the spleen. There is one exception to this rule, however, for the motility of tumors of the stomach is always greater than the motility of tumors of the liver and the spleen if the pylorus itself is adherent to the liver. The differences in the motility of the different organs are merely differences of degree. If the whole stomach is dislocated and if it is no longer in contact with the diaphragm, as in pronounced gastroptosis, the respiratory motility of the organ is lost. If the stomach is merely displaced vertically, it may still possess pronounced respiratory motility. This is clearly demonstrated by the case I have reported above.

The determination of "exspiratory fixation" of the stomach is more important for the diagnosis than the test for the respiratory motility of the organ. Minkowski[1] described this phenomenon for the first time. If a tumor belonging to the stomach, the intestine, or the mesentery is grasped between the fingers and fixed in one position at the height of inspiration, the upward movement of the tumor on expiration can be prevented. If the tumor of the stomach is adherent to the liver, this is impossible, nor is it possible in tumors that belong to the liver or the spleen. If the whole stomach is dislocated, it is particularly easy to prevent respiratory upward movement of the tumor by fixation.

The position that the tumor occupies when the stomach is filled to different degrees is particularly important. If patients with a tumor of the stomach are examined both when the stomach is empty and when it is artificially inflated, marked differences in the position of the tumor will be discovered. As soon as the gas or the air is allowed to escape, the tumor will drop back into its former position. The movements that the growth makes during and after inflation will depend altogether on its seat.

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[1] *Berlin. klin. Wochenschr.*, 1888, No. 31.

Tumors of the pylorus on inflation of the stomach usually move downward and to the right; less frequently upward and to the right. The movements of tumors situated at the lesser curvature vary. Occasionally the whole stomach moves downward when it is inflated. In instances of this kind tumors of the lesser curvature also move downward, and the entrance of the swelling into the gastric region can frequently be determined. As a rule, however, tumors of the lesser curvature disappear backward and upward when the stomach is inflated, provided they are not adherent to the liver. This dislocation backward and upward is due to the fact that distention of the stomach with gas causes the greater curvature to bulge forward. A knowledge of this phenomenon may occasionally enable us to differentiate tumors of the stomach from tumors of the left lobe of the liver, for the latter are usually forced against the anterior abdominal wall when the stomach is inflated, whereas the former, as I have said, are forced in an opposite direction. Tumors of the posterior wall of the stomach that are accessible to palpation when the stomach is empty may disappear after inflation of the organ. Tumors of the anterior wall of the stomach and of the greater curvature usually become dislocated downward on inflation, and occasionally become less accessible to palpation.

This locomotion of tumors according to the state of distention of the stomach is important not only for the diagnosis of the location of the tumor, but also for determining whether or not adhesions exist. The latter point is of great practical importance, for the motility of the tumor demonstrates at once that widespread and solid adhesions have not formed. This is particularly important whenever surgical interference is contemplated. The importance of this method of examining the stomach both when it is empty and when it is distended with air has not yet, it appears to me, been sufficiently recognized, nor is it employed as universally as it should be. In doubtful cases inflation of the colon may be performed, as this procedure may furnish valuable information. If this adjuvant to the diagnosis were employed in all cases of abdominal tumor in which the location of the tumor is undetermined, fewer mistakes in the localization of the tumor would be made, and tumors of the stomach would not so frequently be mistaken for tumors of other organs. Even if it is firmly established that the swelling felt is a tumor of the stomach, inflation should still be performed, for no other manipulation gives us better information in regard to the form, size, and position of the stomach and the motility of the tumor.

Errors in the opposite direction are also frequent. The head of the normal or diseased pancreas has frequently been mistaken for a tumor of the stomach. It is possible frequently to feel the pancreas in thin subjects with relaxed and flaccid abdominal walls. Even though no tumor is present in the pancreas, the organ can occasionally be palpated in the epigastric region. If dyspeptic disturbances are present at the same time, the pancreas, on superficial examination, may be mistaken for a tumor of the stomach. If the stomach is inflated, provided, of course, the organ is in its normal position, the tumor disappears completely. This disappearance is not, however, an absolute proof that the tumor does not belong to the stomach, for tumors of the posterior wall or the lesser curvature of the stomach may also disappear on inflation. If, at the same time, the stomach should be dislocated downward, the possibility of error is still greater. I observed a case of this kind about three years ago.

This patient, a merchant of fifty-four years, developed gastric disturbances and frequent attacks of diarrhea several months before I saw him. As a result of these disturbances his general nutrition became very much impaired. In the beginning all the symptoms he complained of were attributed to a simple gastro-intestinal catarrh. After a short time, however, a tumor that was believed to belong to the stomach was discovered in the epigastric region. At the same time it was found that the lower boundary of the stomach extended below the umbilical line, so that ectasy of the stomach was diagnosed. The diagnosis, then, was carcinoma of the stomach with secondary ectasy. Operative removal of the tumor was proposed. The patient gave his consent, but requested that the opinion of a third physician be secured before the operation was performed. For this reason he came to my clinic.

I examined the patient carefully and employed the method of inflation. I found that the lower boundary of the stomach was situated low down below the umbilicus, but that the lesser curvature was also dislocated downward; in other words, that we were not dealing with ectasy, but with gastroptosis. The tumor was situated above the lesser curvature, and had nothing whatever to do with the stomach; it was merely the pancreas that was very accessible to palpation, owing to the emaciation of the patient and the downward dislocation of the stomach. Aspiration of the stomach-contents failed to reveal any motor disturbances or any alterations in the secretion of gastric juice. The operation, of course, was not performed. The patient rapidly recovered, gained twenty pounds in a short time, and has been free from all disturbances up to the present time.

This case demonstrates how easy it is to commit errors in diagnosis unless all the factors of the case are carefully weighed. It also demonstrates how very important it is to employ all the methods of examination that we know of. If artificial inflation had been performed in this case; if the peptic and motor powers of the stomach had been analyzed, the diagnosis "carcinoma of the stomach" would scarcely have been made, or would, at least, have been considered very improbable. Surgical interference, at all events, would certainly not have been recommended.

The pancreas, however, may also be the seat of a tumor, particularly of a carcinoma. Tumors of the pancreas are usually situated low down, are movable, and can usually be distinguished by these points from tumor of the stomach, of the pylorus, and of the lesser curvature, as well as from tumors of the transverse colon. If characteristic stools containing fat and mellituria are present, the diagnosis of disease of the pancreas is, as a rule, easy. Occasionally icterus and symptoms of portal stasis develop in the course of the affliction. A careful analysis of the chemism of the stomach is, of course, very important in each case.

Tumors of other portions of the intestine are rarely confounded with tumors of the stomach, excepting possibly tumors of the transverse colon and the duodenum. In doubtful cases inflation of the stomach and of the colon will enable us to render a decision. Tumors of the transverse colon, moreover, usually constitute an impediment to the passage of the stools, so that the bowel becomes very much distended with fecal matter and gas above the tumor, particularly if the growth is situated in the ascending colon. If the carcinoma of the intestine is adherent to the stomach-wall, its motility may become impaired. In rare instances carcinoma of the intestine has been known to perforate into the stomach. If this happens, the vomit and the stomach-contents that is removed by the sound contain fecal matter. If the perforation is so small that no fecal matter can enter the stomach, the stomach-contents may, nevertheless, have a distinctly fecal odor. A case of this kind was reported by Leube. In all cases of this character the final decision will be based on an analysis of the stomach-contents.

The differential diagnosis between carcinoma of the stomach and carcinoma of the duodenum is more difficult. Tumors of the latter viscus are frequently indistinguishable from carcinoma of the stomach. I called attention to this fact some time ago, and in order to illustrate this more clearly I append a case-history that I have already published in another place:

M. M., widow, age fifty, began, at about Easter, 1885, to complain of symptoms that seemed to be due to the presence of gall-stones. Since that time she complained of loss of appetite and slight pain in the region of the right costal arch. Later occasional attacks of vomiting occurred. The patient became very much emaciated, and finally entered the clinic on September 28, 1885.

On examination the patient was found to be very much emaciated and cachectic. The liver was enlarged, and its lower margin hard and nodular. In a location corresponding to the incisura a very hard, nodular, pear-shaped tumor was felt at the lower margin of the liver, that merged directly into the greater curvature of the stomach. The stomach was greatly distended, and a distinct succussion-sound could be elicited over the organ. Icterus soon supervened. Repeated attacks of vomiting occurred, during which coffee-ground material was raised. The stomach-contents was repeatedly analyzed, and numerous coarse, undigested morsels of food, in particular coarse meat-fibers, were discovered on each occasion. The filtrate of the stomach-contents gave no reactions for free hydrochloric acid, but a very distinct reaction for lactic acid. The patient died on October 28th.

The clinical diagnosis of carcinoma of the liver, probably starting from the gall-bladder, carcinoma of the pylorus, and ectasy of the stomach was made. The advanced degree of ectasy, the loss of peptic power of the gastric juice, and the vomiting of coffee-ground material all indicated carcinoma of the stomach.

On autopsy a carcinoma of the gall-bladder and liver, perforation of the carcinoma into the duodenum, compression and stenosis of the duodenum by the carcinoma, carcinoma of the portal and retroperitoneal glands, and general icterus were found.

A careful examination showed that the mucous lining of the duodenum had been destroyed in that portion of the intestine where the carcinoma protruded into the duodenum, so that this portion of the intestine was converted into a carcinoma with superficial ulceration. The larger nodule that compressed the descending portion of the duodenum like a cylinder also consisted of masses of cylindric epithelium that had undergone fatty degeneration. The pyloric ring was found open. No evidence of carcinoma was seen in the stomach. The details of this case may be studied from the illustration (see Fig. 29).

It is hardly necessary to give my reasons for assuming the presence of a carcinoma of the stomach in this case. The coffee-ground material that was vomited undoubtedly came from the ulcerated carcinoma in the first portion of the duodenum. This case is particularly interesting in one respect, for it shows that under certain circumstances carcinoma of the duodenum may be made responsible for the absence of free hydrochloric acid in the stomach. The dilatation of the stomach is readily explained from the narrowing of the duodenum. The lumen of the intestine was a mere slit in this case, and naturally acted in the same way as a stenosis of the pylorus.

This case may illustrate the difficulties encountered in rendering a differential diagnosis between carcinoma of the stomach and the duodenum ; it also shows that ectasy of the stomach, and even coffee-ground vomit, although they are considered valuable symptoms in the diagnosis of carcinoma of the stomach, may, under certain circumstances, also be seen in carcinoma of the duodenum.

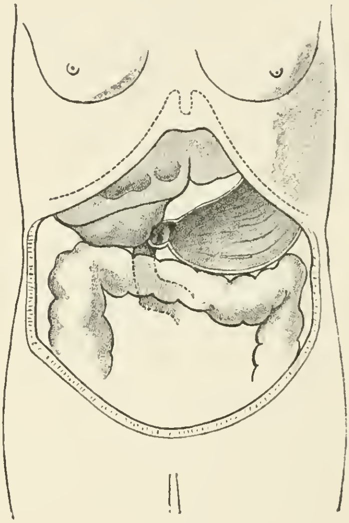

FIG. 29.

I remember another interesting case that I observed some years ago in which all the clinical symptoms of cancer of the stomach appeared,—advanced ectasy, absence of free hydrochloric acid,—but in which autopsy revealed the presence of a carcinoma in the upper portion of the jejunum, with secondary dilatation of the duodenum and the stomach.

The determination of free hydrochloric acid in the stomach-contents should, as Leube says, enable us to render a decision in cases of this kind. The cases, however, that I have just described show that this is not necessarily the case in all patients, for the loss of the peptic power of the gastric juice may, for instance, be due to regurgitation of bile if the carcinoma is located below the papilla, or it may be due to atrophy of the gastric mucosa. If constant regurgitation of bile can be demonstrated in any given case, this feature may be utilized in the diagnosis of infrapapillary carcinoma.

Occasionally carcinoma of the gall-bladder may be confounded with carcinoma of the pylorus. This is illustrated by the case I have just reported. The position of a tumor of this organ is so similar to the position of tumors of the stomach that this point alone may lead to error. If the patients are examined carefully, however, it will, as a rule, be possible to differentiate between the two. Carcinomata of the gall-bladder are, of course, situated exactly in the location of the gall-bladder; they follow the movements of the liver, show no lateral motility, and do not allow exspiratory fixation. When the stomach is inflated, their position is not changed. It may, of course, happen that a tumor of the pylorus becomes adherent to the liver and shows the same peculiarities. Carcinomata of the gall-bladder, however, if they are uncomplicated, rarely produce serious dyspeptic disturbances, nor do they lead to dilatation of the stomach unless abnormal adhesions form that exercise traction on the pyloric portion of the stomach and produce stenosis of this orifice. Free hydrochloric acid is usually present in the stomach in carcinoma of the gall-bladder, whereas in carcinoma of the stomach it is, as a rule, absent.

Tumors of the spleen and tumors of the kidney should not be confounded with carcinoma of the stomach if ordinary care is exercised. Enlarged lymph-glands situated near the ascending aorta may, however, lead to error, particularly if the glands in the radix mesenterii are enlarged. If dyspeptic symptoms are presented at the same time, the diagnosis of carcinoma may seem very probable; if these patients, however, are examined carefully; if, in particular, the stomach is inflated, its motility tested, its contents analyzed, the differential diagnosis is usually easy. Aneurysm of the abdominal aorta should be even less misleading, and should not be confounded with carcinoma of the stomach. This error could be committed only if pulsations from the aorta were transmitted to the tumor of the stomach. As a rule, however, it can easily be determined whether we are dealing with a pulsation that is transmitted to some tumor or with a tumor that performs independent pulsations, for in the former instance the pulsating movements are felt in one direction only—namely, postero-anteriorly, whereas in the latter instance the pulsation is expansile and is felt in all directions. If the subjective symptoms of the patient are studied carefully; if the course of the disease is considered; if the peptic powers of the stomach and numerous other factors that we have enumerated are studied, the two diseases should not be confounded.

Carcinomata of the mesentery and the peritoneum or sacculated peritoneal exudates may occasionally be confusing. Inflation rarely enables us to arrive at definite conclusions, because tumors of the mesentery are usually dislocated downward in the same way as tumors of the stomach. Inflation of the intestine also causes tumors of the mesentery to move downward, and at the same time forces them anteriorly against the abdominal wall. At the same time inflation gives us some valuable information, because this procedure renders the boundaries of the stomach more distinct, so that the organ can frequently be differentiated from the tumor. Examination of the stomach-contents also aids us in making a differential diagnosis between these diseases. Tumors of the mesentery and growths of the peritoneum are usually more diffusely distributed than carcinomata of the stomach, and are rarely movable on respiration. In general they cannot be moved about in the abdomen. It is frequently very difficult to decide whether or not carcinoma of the stomach is present in addition to cancerous nodules that may be felt in the mesentery or the peritoneum. If in cases of this character a tumor of the stomach cannot be felt, the only method at our disposal is to analyze the stomach-contents and to test for free hydrochloric acid. If the latter is found present, the diagnosis of cancer of the stomach is rendered very improbable.

Abscesses of the abdominal walls can be confounded with carcinomata of the stomach only if a very superficial examination is made. The same applies to subserous lipomata and epigastric hernia.

Encapsulated foreign bodies—gastroliths—are said to have simulated malignant growths of the stomach in several instances. In cases of this kind the history of the case and the analysis of the stomach-contents should always enable us to demonstrate that the swelling in the stomach is not malignant.

The presence of a tumor in the stomach does not necessarily indicate that it is a carcinoma; the only way in which to determine this point is to use all the methods at our disposal—namely, to determine the secretion of gastric juice, to study the course and the development of the symptoms, to determine the presence of cachexia, and other factors.

Tumors that are not carcinomatous are sometimes found in the wall of the stomach—for instance, fibroma, sarcoma, myoma, lipoma, adenoma, etc. All in all, tumors of this character are rare. Usually they produce no symptoms whatever. [Exception should be made in the case of sarcoma.—Ed.] Small fibromata may occasionally lead to a narrowing of the lumen of the stomach, or may produce gastritic symptoms. Neoplasms that are not carcinomatous can be diagnosed only if a tumor can be felt. It is almost impossible, however, to determine the nature of the swelling. This is possible only under exceptional and particularly favorable circumstances.

Occasionally the base of an old gastric ulcer will become thickened and converted into a plate-shaped swelling with hard margins. Such a pathologic swelling can occasionally be felt and may simulate a carcinoma. I do not think, however, that carcinoma will frequently be confounded with an old ulcer of this character, for the history of the case, the course of the disease, and the analysis of the stomach-contents will always enable us to make a differential diagnosis without much difficulty.

Functional hypertrophy of the musculature of the pyloric region is more important. Leube states that this condition of the musculature is less frequently encountered than we are usually led to believe, and I agree with him. Hypertrophy of the pyloric musculature rarely leads to the formation of a swelling that can be palpated from without. It is most frequently seen in gastric ulcer, and in hyperacidity that is complicated by violent attacks of cardialgia. Here, too, the anamnesis, the course, and the duration of the disease, the general health of the patient, and a study of the gastric secretion will save us from error. Rosenheim[1] has recently reported a case which shows that under certain circumstances hypertrophy of the pyloric musculature may lead to error, even if all the factors that we have enumerated are considered.

His patient was a woman of fifty-eight years, who had been healthy up to the time she consulted him. In March, 1893, she began to complain of gastric symptoms that gradually increased in severity until they became very distressing. The chief symptoms were loss of appetite, nausea, and vomiting. The patient emaciated rapidly, and was forced to go to bed two weeks after the onset of the symptoms.

When she entered Senator's clinic the stomach was found to be dislocated and slightly dilated. There was a painful tumor of moderate size in the pyloric portion of the stomach. Hydrochloric acid was absent; the lactic acid reaction, made according to Uffelmann's method, was positive. Death occurred four months after the onset of the disease. Virchow performed the autopsy and found no trace of carcinoma. A tumor had been simulated by the hypertrophied muscularis, for the muscular layer was found to be from 0.6 to 0.5 cm. thick. At the same time there was an advanced degree of atrophy of the mucous lining.

The most conspicuous features in this case are, in the first place, the rapid course of the disease; in the second place, the great muscular hypertrophy of the pylorus that simulated a tumor. This hypertrophy was taken for a carcinoma, chiefly because other symptoms that indicated carcinoma were present—namely, rapid emaciation, ectasy, loss of hydrochloric acid secretion, and a positive reaction for lactic acid. As a matter of fact, I can think of no way in which error could have been avoided where a symptom-complex of this kind was presented. Of course, cases of this kind are exceedingly rare, and personally I do not recall a single case in which the totality of the symptoms corresponded so completely to the symptoms of carcinoma of the pylorus. In the great majority of cases, particularly if the patients are observed for a sufficiently long time, a number of points will be discovered that will enable us to differentiate hypertrophy of the muscularis from carcinoma.

Another way in which neoplasms can be simulated is the following: Ulcers of the stomach situated in the anterior wall of the organ may penetrate to the serosa and lead to the formation of an encapsulated exudate in this location. In general, however, the course of the disease, the occurrence of secondary peritonitis or of signs of circumscribed perforation, will enable us to render a diagnosis of ulcer without difficulty. In addition the tumor will be found to be immotile, and fever will usually be present, particularly if adhesions form with the abdominal wall or if abscesses form in the abdominal muscles.

The only way in which the diagnosis of carcinoma on the basis of an ulcer scar can be made is to study the anamnesis, and to determine whether or not symptoms of ulcer were present for a long time. In addition the production of hydrochloric acid will be normal or abnormally increased even though a tumor is present; finally it must be determined that the tumor continues to grow and that cachexia at the same time increases. In many cases of this kind it is possible to determine the gradual transition of hyperacidity into subacidity, and finally anacidity, while at the same time the tumor is seen to increase in size.

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[1] *Münch. med. Wochenschr.*, 1894, No. 29.

So much about the differential diagnosis. I have described the most important points that have to be considered, without, of course, exhausting all the possibilities. Every clinician who has to examine many patients will occasionally encounter one that presents new and unsuspected difficulties.

Prognosis.—The prognosis of carcinoma is usually bad. Internal remedies may palliate certain symptoms, may relieve the distress of the patient, may arrest the loss of strength, but can never cure the disease. Physical methods of treatment, like lavage of the stomach, are only useful in counteracting certain symptoms. The chief task of internal medicine must be to perfect the methods of diagnosis, so that it may become possible to recognize carcinoma of the stomach as early as possible. This disease can only be cured if the diagnosis is made very early, and even then the only method of treatment is surgical. In the majority of cases even operative interference produces merely an improvement in the condition of the patient, but no real cure of the disease. The sooner the disease is recognized the greater the likelihood of success from surgery.

Treatment.—Carcinoma of the stomach can be cured only by surgical means. The results obtained so far from operative interference can hardly be considered particularly favorable. This may be due possibly to poor methods of operation that were chosen in individual cases or to the inexperience of different operators. The chief reason, however, is undoubtedly that the majority of cases are *a priori* unsuited for surgical treatment. In many instances we encounter carcinoma in advanced stages; in others metastases are formed; in still others there are adhesions, or the loss of strength is very great. If we are to expect better results from surgery, operations for carcinoma of the stomach must be performed as early as possible—that is, when the disease is in its earliest incipiency. In order that this may be done, internists must make the diagnosis very early, before the typical picture of carcinoma, with pronounced cachexia, a distinctly palpable tumor, advanced ectasy, etc., has developed. Only when patients with carcinoma of the stomach are turned over to the surgeon at the very onset of the disease can we expect to see better results from operative measures. Unfortunately, physicians in clinics and large hospitals are usually unable to make early diagnoses for the reason chiefly that patients who enter institutions of this kind present themselves in advanced stages of the disease; consequently we can hardly expect to see the statistics in regard to the operative treatment of carcinoma of the stomach very much improved in clinics and hospitals. The general practitioner, however, frequently sees cases at an early stage, and it devolves upon him to improve our record in this field.

Before discussing the question of operative treatment of carcinoma of the stomach I will briefly sketch the methods that are at the disposal of the internist for treating this disease. The methods of treatment will, of course, vary according to the seat of the carcinoma, according to the motor powers of the stomach, and numerous other circumstances. Certain therapeutic postulates, however, must be fulfilled in all cases. The nutrition of the patient, for instance, must be raised as much as possible in all cases, the diet must be regulated to correspond to the functions of the stomach.

The internist possesses three means for treating carcinoma of the stomach—namely, first, medicamentous measures; second, mechanical methods; and third, dietetic regulations.

Medicinal treatment, as we have said above, is only indicated in the treatment of certain symptoms, although many investigators at different times have claimed to cure by drugs. Many remedies have been recommended in the course of time as specifics for carcinoma of the stomach, but none of them has maintained its reputation. For this reason I will refrain from enumerating any of these drugs, as it is of no interest nowadays to know what was once claimed for them. I will only briefly mention condurango bark, that was recommended as a specific for carcinoma of the stomach by one of the most eminent clinicians, Friedreich.[1] Unfortunately, Friedreich's statements were found to be incorrect; I[2] repeated many of his investigations and administered the drug to many cases of carcinoma of the stomach without discovering any specific action. In some of these cases a favorable effect was undoubtedly noticed, for the appetite improved and the general strength of the patients increased, but beyond this no good results were seen. At the time I expressed the opinion that condurango bark possesses considerable stomachic powers, but that it has no virtues as a specific. I still adhere to this view to-day, and I believe that the majority of investigators agree with me. Riess[3] is the only one who still praises this remedy extravagantly and claims wonderful things from it in the treatment of carcinoma of the stomach. It would lead us too far to enter into the detailed discussion of Riess's observations; I must say that they are not convincing and do not justify us in assuming that the drug has any specific powers. Condurango can undoubtedly improve the appetite. This observation has frequently been made, but neither I nor anybody else has even seen permanent improvement or a cure of cancer of the stomach from the exhibition of this drug. Condurango bark, therefore, is merely a symptomatic remedy and acts by virtue of its stomachic powers. As a stomachic, therefore, I certainly recommend the drug. At the same time it must be remembered that even this power of the drug may fail, particularly in those cases in which the secretory and motor powers of the stomach are almost completely lost. I have observed that the drug is efficient only in the earlier stages of carcinoma—that is, at a period of the disease when the motor power of the stomach is not greatly impaired.

Condurango bark may be given in different forms. It is most frequently administered as the decoction of the maceration. I am in the habit of administering the drug as follows:

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[1] *Berlin. klin. Wochenschr.*, 1874. [2] *Ibid.*, 1874. [3] *Ibid.*, 1887.

℞.	Cort. condurango,	18.0 — 20.0;
	Macera per horas duodecim c. aq.,	300.0;
	digere leni calore ad col., adde	180.0;
	Acidi hydrochlor.,	1.5.

M. D. S. A tablespoonful three to four times a day before eating.

The addition of syrups is not to be recommended. The small quantity of hydrochloric acid that I have added to this prescription is naturally not intended to cover the deficit of hydrochloric acid in the gastric juice; it is merely administered as a stomachic, for we have seen that hydrochloric acid may increase the appetite in the same way as condurango bark. The drug is also frequently administered as condurango wine, and occasionally in the form of the very expensive fluid extract of condurango.

Penzoldt has recommended basic orexin (dose about 0.3 gm. or 5 gr.), and advises using this remedy in the treatment of carcinoma. I have failed to see any appreciable results from the exhibition of this drug in carcinoma of the stomach. Some of the other well-known stomachics may also be employed. Kussmaul and Fleiner have recommended irrigation of the stomach with infusions of hops and quassia wood, and claim that this treatment stimulates the appetite. This method, of course, acts merely as any stomachic would if administered by mouth, and at the same time aids in thoroughly cleansing the stomach. Lavage of the stomach alone occasionally exercises a stomachic effect in addition to cleansing the stomach.

All other methods that have been recommended in the treatment of carcinoma of the stomach are only useful in the treatment of certain symptoms. What has been said of the above-mentioned remedies applies with equal force to the whole galaxy of remedies that are used in this disease. There are many cases in which medicamentous treatment is altogether superfluous, and in which a careful diet combined with lavage of the stomach is all that is needed.

The best method for combating vomiting, a symptom that is seen with particular frequency in carcinomatous stenosis of the pylorus with secondary ectasy, is methodic lavage. Vomiting can almost always be stopped by this method. If stagnation of the stomach-contents occurs, so that fermentation- and decomposition-products are formed, certain antifermentative remedies, as salicylic acid, resorcin, or boric acid, may be added to the wash-water. Even in those cases in which vomiting is not the result of prolonged stagnation of stomach-contents, but is merely due to an abnormal irritability of the gastric mucosa, lavage may be useful. In cases of this kind ice pills, champagne, carbonated waters, morphin, belladonna, and similar remedies may be tried. Narcotics, however, had better be avoided altogether; if they seem indispensable, they should be administered subcutaneously or *per rectum*. If vomiting persists despite of all these measures, the stomach should be placed absolutely at rest for several days and alimentation be carried on by rectal enemata.

Hematemesis may occasionally call for the exhibition of drugs. As a rule, however, gastric hemorrhage is slight in carcinoma of the stomach, and can usually be combated with dietetic or physical means. Hot food or drink, of course, should be interdicted. The patient should keep perfectly still and remain in bed even if the hemorrhages are very slight. The diet should be limited to liquid articles of food, as milk, meat broth, etc., and all solid food should be avoided. The best method, of course, is to place the stomach completely at rest for several days. Remedies that are administered internally are of little value. Chlorid of iron is often recommended, but it is hardly sufficiently concentrated, at least in the form in which we give it internally, to possess any hemostatic action. Secale cornutum, ergotin, and acetate of lead may all be tried if the hemorrhage is severe. [Adrenalin chlorid solution is of undoubted value in relieving these slight hemorrhages.—Ed.] Better than all these drugs, however, is complete rest of the stomach as soon as the hemorrhage becomes severe. For several days no food should be given by mouth, and all nourishment shall be administered in the form of nutritive or liquid enemata in the manner described above (see General Part, page 209). In severe hemorrhages a small ice-bag or a cold compress may, of course, be applied to the region of the stomach. This procedure is probably quite useful. Under certain circumstances opiates may be given in the form of suppositories in order to arrest the peristaltic movements of the stomach and intestine.

The pain in carcinoma rarely becomes so severe as to require special treatment. Priessnitz compresses in the region of the stomach, warm poultices, a Japanese warming-box, and similar measures usually suffice to stop the pain. The attacks of pain frequently occur at certain times only, and seem to be dependent on the retention of ingesta and the distention of the stomach that results therefrom. In these cases aspiration of the stomach-contents is much more efficient in stopping the pain than any narcotic. In certain cases, however, narcotics will have to be given. Boas recommends administering chloroform, three to five drops on ice, in such cases. He speaks less favorably of the chloroform water that is usually given. If possible, the exhibition of narcotics proper should be postponed as long as possible, for opiates have the great disadvantage of increasing any tendency to constipation that may exist. This applies less to morphin. I prefer giving belladonna in these cases. I never administer chloral hydrate, even though its effects are usually good, to patients who are very weak and much reduced, for the reason chiefly that even relatively small doses may exercise a weakening influence on the heart. Codein deserves a trial. If the attacks of pain become very violent, subcutaneous injections of morphin with the addition of atropin are the most suitable remedy.

If the attacks of pain are due to a complicating circumscribed peritonitis, this condition should be treated according to the established rules for treating peritonitis. Rest of the intestinal tract is the first postulate, so that the administration of opium and the application of cold should be tried.

Disturbances of the intestinal function of two forms may occur in carcinoma. We have already called attention to the fact in discussing the symptomatology of this disease that in one series of cases there is more or less obstinate constipation, in a second series frequent diarrhea, in a third series normal and spontaneous evacuation of feces. If constipation is not very obstinate, simple irrigation of the intestine with warm water, possibly with the addition of a little soap, etc., is sufficient. The injections, however, should be high, and the irrigation of the bowel should be performed daily at the same hour, not at different times on successive days and not at intervals of several days. Injections of oil, glycerin suppositories, and injections of glycerin may occasionally be useful, particularly if there has been prolonged stagnation of fecal matter in the lower portion of the colon. Provided the diet and the mode of life are carefully regulated, these measures will, as a rule, suffice, so that the administration of laxatives or purgatives becomes unnecessary. If these simpler measures fail, however, rheum, aloes, tamarind, the fluid extract of cascara sagrada, and similar remedies, may be tried. Saline purgatives and the drastic remedies should not be employed.

The best method of treating diarrhea in these cases is carefully to regulate the diet and to perform lavage of the stomach. I have already mentioned that diarrhea supervenes particularly in those patients who are still assimilating a certain amount of food. The primary cause of these attacks of diarrhea is probably insufficiency of the gastric function, leading to disturbances in the normal conversion of food and the formation of abnormal products of digestion. If these old and decomposed remnants of food are removed from the stomach before new food is introduced, and if the diet is adapted to the digestive powers of the stomach, it will frequently be possible to stop the diarrhea, or at least to improve it very much by these simple measures. If this method of treatment should fail, antiseptic drugs, like salol, benzonaphthol, salicylate of bismuth, or dermatol, should be given. Astringent remedies are less suitable. Under certain conditions antiseptics may be given together with opiates.

Other complications of carcinoma of the stomach may occur at any time in the course of the disease, and may naturally call for treatment or palliation by other remedies. If perforation, for instance, suddenly occurs, and if it is impossible to perform laparotomy at once, ice and opiates, the latter preferably in the form of suppositories, should be given. If marantic hydrops supervenes, heart tonics, like digitalis or camphor, may be indicated. If advanced degrees of ascites are present, paracentesis of the abdomen may become necessary.

We have no specific remedy for the treatment of coma carcinomatosum. All we can do for these cases is to administer stimulants.

It would be useless to enumerate all the other remedies that have been recommended for the treatment of cancer of the stomach, or that might possibly be considered. I may only mention that Boas[1] claims to have seen good results from the administration of iodids and from arsenic. He particularly recommends the former, and states that they act as a good palliative both in carcinomata of the esophagus and of the cardia. I have frequently employed iodids in carcinomata of the esophagus and the cardia, but have never seen any appreciable effects. Maibaum[2] reports much improvement in 2 cases of carcinoma of the stomach with tumor from the administration of pyoktanin in pill-form (0.06 three times a day), or in suppository (0.06 a dose) with extract of belladonna (0.02). Fay[3] recommends the sulphate of anilin (0.1 to 0.8, slowly increased), and Brissaud[4] speaks in favor of sodium chlorid.

In conclusion, I will add a few words on the employment of hydrochloric acid and of ferments. The suggestion seems natural to administer hydrochloric acid in those cases in which the secretion of hydrochloric acid is reduced, and to attempt to improve the digestion of proteids in this way. In regard to the feasibility of doing this I refer to what has been said in the General Part of this work (page 227). The digestion of albumin is rarely improved by the administration of hydrochloric acid, even if large doses are given together with pepsin. This is due to the fact that the quantity of hydrochloric acid that we can administer in this way is very much smaller than the quantity needed to make up the deficit of hydrochloric acid in the gastric juice. I also refer to what has been said above in regard to papayotin, papain, and pancreatin. If the motor power of the stomach is still good, these adjuvants to digestion are superfluous, because the intestine assumes the functions of gastric digestion. In cases with advanced atony of the gastric mucosa it is much more important to accelerate the evacuation of the stomach-contents into the intestine. The administration of digestive ferments in these cases is altogether insufficient to improve disturbed gastric digestion and to compensate the deficiency of ferments in the gastric juice. At the same time papain[5] may be tried in cases of subacidity or anacidity.

The second method of treatment that the internist must consider in carcinoma of the stomach is mechanical treatment. This is particularly applicable to those cases in which a carcinoma is situated at one of the orifices of the stomach and produces stenosis.

Carcinomata of the cardia that cause narrowing of the cardiac orifice and the lower portion of the esophagus may call for the application of sounds or bougies. As long as the passage is more or less open the passage of sounds is not indicated. In the early stages of this disease we frequently find that the sound seems to pass the cardiac region with some difficulty; if this occurs, and if small bloody particles are occasionally found in the openings of the sound, we should always be suspicious of carcinoma. Even under these conditions a course of sounding is not necessary; on the contrary, it had best be avoided, as the irritation of the parts may lead to an exacerbation of the disease-process.

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[1] *Diagnostik u. Therapie der Magenkrankh.*, 1896.
[2] *Petersburg. med. Wochenschr.*, 1894, No. 4.
[3] *Deutsche Medicinal-Zeitung*, 1894, No. 67.
[4] *Jour. de méd. de Paris*, 1894, No. 47.
[5] Compare Grote, *Deutsche med. Wochenschr.*, 1896, No. 19.

If, on the other hand, a severe degree of stenosis has developed in the cardiac region, we should begin a course of sounding. There is a great diversity of opinion in regard to the frequency with which sounds should be passed in carcinomata of the cardia that produce stenosis. I do not deny that an unskilful and careless manipulator may do a great deal of damage with the sound. If the instrument, however, is introduced delicately and carefully, and if sounds of a suitable caliber are selected, I do not see how any harm can be done; on the contrary, to judge from my personal experience, improvement nearly always follows. Patients who were altogether unable to introduce any food into the stomach soon became able to swallow their food. If I had the choice in any given case between passing the sound carefully and at frequent intervals and not passing the sound at all, I should certainly prefer the former method of treatment. I refer for the details of this question to the section on Narrowing of the Esophagus.

Whereas the advisability of treating carcinoma of the cardia with sounds and bougies is questioned by some, there is a great uniformity of opinion in regard to the advisability of treating by mechanical means advanced stenoses of the pylorus that have led to severe degrees of secondary ectasy of the stomach. All investigators seem to agree that this disease is a favorable one for mechanical treatment, and all advise washing out the stomach with the aid of the stomach-tube. Patients of this kind feel immensely relieved after the first lavage—the pain seems to disappear as if by magic. The same applies to the attacks of vomiting, that may have been exceedingly obstinate up to that time. Lavage of this kind, however, is indicated not only in those cases in which the ectatic organ, so to say, overflows spontaneously from time to time, but also in those instances in which there is a considerable degree of ectasy without frequent vomiting.

In advising lavage of the stomach we should not be governed by the size of the organ, but merely by the degree of motor insufficiency. The stomach may be of abnormal size and still possess sufficient and altogether normal motor powers; inversely the stomach may be of normal size and still be insufficient in regard to its motor function. If the stomach contains numerous coarse particles of food six or seven hours after a test-meal or one or two hours after a test-breakfast, this may be considered a positive sign of motor insufficiency. A practical method for determining the degree of motor insufficiency, particularly in those cases in which the stomach is unable to get rid of the ingesta within the normal time after a test-meal or a test-breakfast, is to wash out the stomach in the evening, then to administer a simple supper, and to remove the stomach-contents again the next morning before breakfast. If an abundant quantity of undigested material or of other residue is found at this time, we may know that the patient is afflicted with an advanced degree of motor insufficiency; if, however, the stomach is found empty in the morning, whereas it contained coarse particles of food six or seven hours after a test-meal, we know that such a patient is afflicted with a less severe degree of motor insufficiency than the first one.

Methodic lavage should be instituted in all cases where the period of digestion is found to be considerably prolonged.

In carcinoma this motor insufficiency is, as a rule, coupled with a more or less advanced degree of secretory insufficiency. Consequently we see not only stagnation of stomach-contents, but also abnormal fermentation and decomposition, and as a result of all this vomiting, and later disturbances of appetite. Methodic lavage not only stops the vomiting and the abnormal decomposition-processes that are going on in the stomach, but may even increase the appetite, and in this way aid in raising the general nutrition of the patient.

The question arises, When shall we aspirate the stomach-contents in carcinoma of the pylorus? I believe that the best time is in the evening before supper. If numerous coarse, undigested particles of food that are undergoing putrefaction and fermentation are present in the stomach at the time when the patient eats his supper, it is certain that they do harm, and it is rational to remove this material from the stomach before new material is introduced. If, in addition, it is found that a portion of the food that is introduced in the evening, after lavage of the stomach, is present in the stomach the next morning, then the stomach should be washed out again in the morning, for the presence of portions of the supper early in the morning show that gastric digestion is greatly impaired, and that all the food has not been propelled into the intestine within the normal time.

Some clinicians have objected that lavage performed both morning and evening may remove much nutritive material. That in a certain sense is true, and lavage in these cases must be considered wasteful and a luxury, but it is better not to introduce new food into a diseased stomach until the fermenting and putrefying remnants that it contains are removed. If the organ is thoroughly cleansed and a light and easily digestible diet administered, more will be absorbed in the end than if new food is given on top of old, undigested and decomposed ingesta. If the motor power of the stomach is intact, lavage is not indicated, even though the secretion of gastric juice may be very much perverted. We quite frequently encounter cases in which the ingesta are removed from the stomach within the normal time even though free hydrochloric acid is constantly absent from the gastric juice. Motor insufficiency and atonic ectasy may also be present without stenosis of the pylorus; this is seen with particular frequency in cases of diffuse carcinomata that develop rapidly, and particularly in those cancers that involve the greater curvature. Here, too, of course, lavage is called for.

Massage and electricity play a very insignificant rôle in the treatment of carcinoma cases as compared to methodic lavage. These methods are occasionally very useful in the treatment of atonic ectasy, but they are valueless in the treatment of ectasy of the stomach that is due to stenosis of the pylorus. In the latter cases we are merely dealing with relative motor insufficiency, for the musculature of the stomach is not atonic but, on the contrary, hypertonic. Notwithstanding this hypertonicity or even hypertrophy of the musculature of the stomach-wall, that is manifested by increased and distinctly visible peristaltic movements, the stomach is unable to get rid of its contents within a normal time. If the stomach-contents cannot be evacuated, even though the musculature of the stomach is hypertrophied, we can hardly expect to see favorable results in this respect from the employment of massage or electricity. In that form of atonic ectasy, however, that is occasionally seen in diffuse soft carcinomata that do not involve the orifices of the stomach, a careful trial with electricity may be made in the early stages of the disease. Massage, of course, is distinctly contraindicated even in these cases.

The most important therapeutic measure that we can employ in the treatment of carcinoma of the stomach is, as we have said above, the regulation of the diet. It is impossible to arrange a dietary that would be suitable for all cases of carcinoma, for each case presents certain individual peculiarities. If, for instance, the peptic power of the stomach alone is disturbed, the diet should be different from that used when the motor power of the stomach is also disturbed. Even though the secretory function of the stomach is greatly reduced, the absorption of food need not necessarily be impaired, provided the motor power of the stomach remains intact. If both the peptic and the motor powers are reduced, the absorption of food, of course, will suffer. We see, therefore, that in prescribing a diet we must consider not only the peptic powers of the stomach, but also the state of its motor powers. We must also regulate our diet in such a way that we stimulate the appetite that is frequently impaired in these cases, and must, moreover, consider the violent aversion to meat that many cancer cases develop; in other words, the dietary must be modified in many ways to suit the appetite, the general strength of the patient, the secretory and motor powers of the stomach, etc. We must never forget that in carcinoma we are dealing with conditions different from those found in ulcer. In the latter affection a definite dietary is indicated; we know that all articles of food should be avoided that can irritate the ulcer directly or indirectly. In carcinoma the matter is not so easy, for the cancer *per se* does not call for a definite diet; we merely regulate the diet in these cases according to the sequelæ and complications of cancer that may be present; for instance, we consider certain inflammatory or atrophic states of the gastric mucosa, stenosis of the pylorus, or secondary ectasy of the stomach.

In arranging the diet-list for a cancer case, we should not be too strict. The appetite of the patient, his general habits and his tastes should always be considered. If the patient expresses a desire for certain articles of food, we should satisfy his craving as far as that is possible—that is, we should give him anything he wants to eat unless we know that it is absolutely indigestible, and that it will certainly disagree with him. We can go still further, and say that the patient may eat anything that he knows from experience agrees with him.

As a general rule, the food should fulfil the following conditions: it should tax the stomach as little as possible, it should make small demands on the chemical powers of the stomach and should not cause overloading of the stomach; it should preferably be soft and pultaceous; starchy material, digestible vegetables, leguminous plants, and gelatinous food should constitute the chief diet.

It is best to order the patient to take small meals at frequent intervals, particularly if the appetite is reduced or if the peptic powers are impaired; this applies particularly to those cases in which the motor power of the stomach is reduced or threatens to become so.

If milk can be well borne, it should be given, but only in small quantities. Kefir, kumyss, or peptonized milk usually agrees better than milk. As a rule, the patients do not care for meat; if they take it at all, it should be given in small quantities, preferably finely divided. The diet should consist of meats, particularly white meats, poultry, fish (perch, pickerel, halibut, trout, shad, etc.). Potatoes should only be given mashed, never in a coarse form. Cabbage and black bread should be avoided. Other suitable articles of diet are oatmeal, flour soups, soups or decoctions of tapioca, rice, sago, maizena, leguminose, possibly with the addition of a little peptone or meat solution; spinach, asparagus, cauliflower, turnips, carrots, apple sauce, pear sauce, stewed prunes, chocolate, cocoa, cakes, Zwieback, toast. Light dishes prepared from flour are also usually well borne. Fat, as a rule, does not agree very well with these patients, particularly if it is given in the form of fat meat, fat roast, or fat gravies; the best way in which to give fat is as good butter on cakes, Zwieback, and fine wheat bread (the latter to be used chiefly as toast). Cocoa, butter, and the "Kraft" chocolate recommended by von Mering are also useful in this respect, as they contain very much fat. We should attempt to induce the patient to take some fat in any one of these forms, as this article of diet possesses great caloric value.

Wiel recommends honey or dextrose. I do not think that these two articles should be given where there is a tendency to fermentation. Liquors that ferment easily should also be avoided; claret, brandy with water, port wine, etc., are comparatively well borne. If there is an advanced degree of stenosis of the pylorus with motor insufficiency, the quantity of fluid that is given by mouth should be limited, and large enemata should be administered instead. Tea, cocoa, and coffee are permissible, but beer had better not be taken. At all events it is well to consider the tastes and desires of the patients as far as that is possible.

Many artificial foods have been recommended in place of meat. Chief among them are preparations of albumose and peptone. These articles, however, can never replace meat; in addition they irritate the gastro-intestinal tract if taken in large quantities.

The best artificial food is nutrose or tropon. The patients, as a rule, like to take it, and no bad effects follow its administration. One of the chief advantages of nutrose, therefore, is that it can be given in large quantities. It may be taken in all kinds of soup (one to one and a half tablespoonfuls for each plate of soup), in milk, coffee, cocoa, etc. Blum has recently recommended a preparation called protogen, but this food has not been tried long enough to enable me to make any statements in regard to its utility. According to the investigations of Deucher,[1] protogen is well digested even if the stomach is anacid or stenosed. Deucher also compared it with other albuminous material, and found that more protogen was absorbed than any of the other foods.

It is impossible, therefore, as I have said, to arrange a dietary that is suitable for all cases. In some cases the dietary like the one arranged for the severe form of chronic gastritis and atrophy of the gastric mucosa is called for; in other cases, the dietary must be arranged to suit the condition of ectasy and motor insufficiency that may be present. We should never content ourselves with merely telling the patient to eat nothing that is difficult to digest, and to select a light diet himself. Every case of carcinoma of the stomach should be distinctly told what to eat and what to avoid. If possible, a careful dietary should be written out in each individual case.

The following is an example of a diet-list arranged by Wegele that is suitable for a case of carcinoma without symptoms of advanced ectasy:

	Albumin.	Fat.	Carbohydrate.	Calories.
In the morning:				
150 gm. of maltoleguminose cocoa	6.0	4.0	13.5	
In the forenoon:				
200 gm. of kefir	6.0	4.5	3.8	1.0
At noon:				
150 gm. of maltoleguminose soup	4.0	0.15	9.3	
100 gm. of scraped beefsteak	20.0	6.0		
In the afternoon:				
150 gm. of maltoleguminose cocoa	6.0	4.0	13.5	
In the evening:				
100 gm. of scraped ham	25.0	8.0		
150 gm. of tapioca mush	7.0	5.0	8.0	
10 P. M.:				
200 gm. of kefir	6.0	4.5	3.8	1.0
Addition to cocoa:				
30 gm. of honey	0.4	. .	22.0	
Addition to kefir:				
20 gm. of brandy	. .	. .	. .	14.0
In the course of the day:				
50 gm. of Zwieback	6.6	1.0	35.0	
	87.6	37.1	108.9	16.0
Total caloric value about	1250 calories.			

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[1] *Berlin. klin. Wochenschr.*, 1896, No. 48.

The following is another dietary that I copy from the cook-book of Biedert that I have so frequently referred to (this dietary, too, is for a patient without serious motor disturbances of the stomach):

	Albumin.	Fat.	Carbohydrate.
6 A. M.:			
500 gm. of milk, 40 gm. of toast	20.3	18.4	55.8
8 A. M.:			
Oatmeal soup with 15 gm. of meat solution or soup made from ¼ Timpe soup lozenge	5.5	1.0	14.2
10 A. M.:			
Cream mixture ("a" [1]), 40 gm. of toast	7.8	12.9	41.5
12 M.:			
(*a*) Barley soup with yolk of 1 egg	4.0	9.2	7.7
(*b*) 140 gm. of roast-beef, venison, poultry, boiled chopped beef or fish	42.8	10.4	
(*c*) 40 gm. of toast	3.3	0.4	30.8
(*d*) 25 gm. of cinnamon-cake, soda-cake, coffee-cake, biscuit	2.0	1.5	14.0
(*e*) Small cup of black coffee.			
4 P. M.:			
250 c.c. of milk-water cocoa, 3 Zwieback (30 gm.)	9.2	11.3	38.3
7 P. M.:			
(*a*) Leguminose soup with 15 gm. of meat solution or soup made from ¼ Timpe soup lozenge	7.6	1.0	12.6
(*b*) Rice-flour mush	18.3	14.1	98.1
	120.8	80.2	313.0
Total caloric value about	2524 calories.		

The second dietary is more nourishing than the first one.

	Albumin.	Fat.	Carbohydrate.
6 A. M.:			
250 c.c. of milk, 30 gm. of toast	11.0	9.3	35.6
8 A. M.:			
2 eggs, 20 gm. of toast	13.7	10.2	15.4
10 A. M.:			
125 c.c. of cream, 2 Zwieback	6.9	14.0	18.8
12 M.:			
(*a*) 140 gm. of roast-beef, venison, poultry, boiled chopped beef or fish	42.8	10.4	
(*b*) 40 gm. of toast	3.3	0.4	30.8
(*c*) 25 gm. of soda-cake, cinnamon-cake, coffee-cake, biscuit	2.0	1.5	14.0
4 P. M.:			
250 c.c. of milk-cocoa, 3 Zwieback with fruit jelly	13.5	15.8	44.6
7 P. M.:			
Rice mush, 2 Zwieback, cakes, etc.	14.8	10.8	78.7
10 P. M.:			
250 c.c. of milk, 2 Zwieback	10.9	10.5	26.3
	118.9	82.9	264.2
Total caloric value about	2341 calories.		

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[1] Cream mixture "a" consists of 125 c.c. of cream and 6 gm. of lactose.

The dietary on page 730 is also arranged by Biedert and Langermann, and is suitable for cases in which there is both motor insufficiency and ectasy.

I merely give these few examples, and repeat that modifications must be instituted to suit the peculiarities of each individual case. If the appetite is lost or if the propulsion and absorption of the ingesta become impaired, it is particularly difficult to select a suitable dietary. In the later stages of the disease we must frequently rely upon nutritive enemata.

I need hardly mention that all mental and bodily overexertion should be avoided, and that the patient should be kept as quiet as possible. If the patients remain quiet, general metabolism is greatly reduced, and this is very important when the nutrition of the patient is impaired.

I will now discuss the operative treatment of carcinoma of the stomach. While this method of treatment is the last measure that I recommend, I do not by any means wish to indicate that it is really the last aid, and that it should only be attempted when all other methods have failed. Operative treatment of carcinoma undoubtedly deserves first place. The sooner surgical interference is instituted the better the prospects of recovery. As a matter of fact, permanent good results can only be expected if carcinoma cases undergo surgical treatment very early in the disease; but even in these cases a complete recovery is rare even if we succeed in removing the cancer altogether. A few exceptions, however, are on record in which the patients recovered.

If a carcinoma of the breast or a carcinoma of the lip is removed by operation, the patient is thereby cured. All that remains of the disease is a defect, possibly a disfiguring scar. If a carcinoma of the stomach is removed by operation, the patient, it is true, is no longer carcinomatous, but his stomach-trouble is by no means cured; in other words, the patient no longer suffers from carcinoma of the stomach, but he suffers from other affections of the stomach. This is due to the fact that the atrophic condition of the gastric mucosa that is caused by the carcinoma persists after the tumor is removed. If the secretion of the stomach was perverted and insufficient at the time of the operation (and this is probably the rule excepting in those cases in which carcinoma develops on the basis of an ulcer), the secretory perversions of the organ will persist even after operation. In isolated instances, it is true, free hydrochloric acid has been found in the stomach after operative removal of carcinoma, even though it was absent from the stomach before the carcinoma was removed. Instances of this kind are, however, rare. Almost without exception, cases of carcinoma of the stomach that are fit subjects for operation suffer from a degree of atrophy of the gastric mucosa that is incapable of restitution to normal.

The operation, however, even though it may not improve the secretory powers of the stomach, may lead to very satisfactory results in other directions. Resection of a carcinomatous pylorus frequently leads to apparent recovery of the patients: they gain weight, and may enjoy well-being for a period of years. This is not surprising, for we know nowadays that the food may be well digested even if the secretion of the stomach is greatly reduced, provided the motor power of the stomach is intact, so that the ingesta are completely evacuated into the intestine within a normal time. If this compensatory process does not occur, nutrition naturally suffers. The result of operative removal of carcinoma, therefore, is dependent on two factors: in the first place, the carcinoma must be completely removed; in the second place, the motor function of the stomach must be good—if it was not good before the operation, the operation must restore it to its normal or nearly normal power. If this cannot be accomplished, the nutrition of the patient will remain impaired even though the carcinoma is removed; and these subjects finally perish from insufficient absorption of food and inanition. Occasionally this unfavorable issue is postponed for a considerable time even in these cases.

The surgical procedure that is adopted must consider these two main indications. If for no other reason, the secretory and the motor powers of the stomach should be repeatedly examined and carefully determined in every case in which operative interference is considered. I might mention in this place that operative removal of the pylorus does not necessarily abolish the sphincter action of the pyloric portion of the stomach. Rosenheim has reported a case in which resection of the carcinomatous pylorus did not simply convert the pylorus into hard cicatricial tissue; in this case it seemed as though the musculature in the pyloric region assumed the regulatory function of the pylorus, for if gas or air were forced into the stomach it did not immediately escape into the intestine.

Rosenheim, Kaensche, and others have demonstrated that resection may restore the motor functions of the stomach. They showed this by very careful investigations, but demonstrated at the same time that the secretory functions, as we have mentioned above, are rarely improved by this operation.

The most rational treatment of carcinoma is undoubtedly operative removal—that is, resection of the carcinomatous part. Carcinomata of the pylorus above all are suitable for resection; but tumors in other sections of the stomach may also be amenable to surgical treatment. Carcinomata of the cardia form an exception, for they can probably not be removed by operation. The only surgical procedure that could be employed in treating a lesion in this region would be gastrotomy followed by feeding through a fistula. In the majority of cases this operation has been performed at a very late stage of the disease. If any good results are to be expected, the operation should be performed early. Only then may we expect to see the life of the patient prolonged, for we have shown, in discussing carcinomata of the pylorus, that gastro-enterostomy, by preventing the irritation of the carcinoma by the ingesta that pass over it, arrests the growth and development of the neoplasm to a considerable extent. Witzel,[1] Frank,[2] and others have devised a number of im-

[1] *Centralbl. f. Chirurg.*, 1891, No. 32.

[2] *Wien. klin. Wochenschr.*, 1893, No. 13.

proved methods for performing this operation, so that the prognosis nowadays is much better than formerly. At the same time this method of treatment is not a radical cure; it merely enables the patient to take more food than before, while at the same time the carcinoma remains.

The most favorable conditions for operation are given if the carcinoma is situated at the pylorus. Billroth, as we know, performed resection of a carcinomatous pylorus as early as 1878. Since that time this operation has been performed in numerous cases, in many instances without leading to good results. Surgeons, however, soon recognized that carcinoma of the pylorus is inoperable in many cases. This led to the adoption of another plan—namely, to create a new passage for the ingesta that leads around the stenosed pylorus; in this way it was thought that the condition of the patient could be improved even though the tumor was not resected. The operation that has been devised for this purpose is gastro-enterostomy, or the artificial creation of a passage between the stomach and the jejunum, through which the ingesta can pass into the intestine. Gastro-enterostomy, therefore, is merely a palliative operation. In performing this operation surgeons do not attempt to cure the disease or to remove morbid tissue as in resection, but merely to remove the sequelæ of stenosis of the pylorus. The good results that are frequently seen from gastro-enterostomy in such cases show that the stenosis *per se* and the impediment it constitutes to the passage of the ingesta from the stomach into the intestine play an important rôle in the general loss of strength that is usually seen in this condition. Even if the carcinoma itself is allowed to remain, patients in whom gastro-enterostomy is performed frequently recover to a great extent, and in a very short time after the operation gain in weight, and may even present a healthy, florid appearance. The good results following this operation unfortunately are only transitory. At the same time this operation is indicated in all cases in which complete removal of the carcinoma is no longer possible, and in which symptoms of advanced stagnation of stomach-contents appear.

We may, therefore, say *a priori* that resection of the pylorus is indicated wherever complete removal of the neoplasm is possible. Gastro-enterostomy is indicated wherever the carcinoma cannot be completely extirpated, but where symptoms of advanced stenosis and of stagnation of stomach-contents exist. The gastroduodenal fistula that is created in this operation is intended merely to facilitate the passage of the ingesta into the duodenum. If there is no stagnation of stomach-contents, gastro-enterostomy is useless.

Two objections have been formulated against either operation. Some authors call attention to the fact that the results of these operations are not very brilliant; others have argued that the operation itself is not devoid of danger.

Statistics constructed from the results published by various investigators are of little value in determining the validity of these objections. Even the statistics of one operator fail to decide the matter. Billroth, for instance, reported his experience at the Tenth International

Medical Congress in Berlin, and stated that of 29 cases of resection of the pylorus for carcinoma, he lost 16 as a result of the operation and saved the lives of 13; of 28 cases in which gastro-enterostomy was performed for carcinomatous stricture of the pylorus, 14 died and 14 recovered. Of 57 cases, therefore, that were operated upon, 30 died from the operation and only 29 recovered, consequently the mortality was 52.6 per cent. Hahn, on the other hand, recently reported 15 cases of gastro-enterostomy, three-fourths of which were cases of carcinoma, and this operator did not lose a single case of this series.

The final outcome will depend not only upon the experience and the skill of the operator, but also upon the indications that exist for performing the operation. The results that different operators report cannot be compared unless each case is carefully studied in relation to these two factors. Kraske is right when he argues that cases of this kind must not be counted, but judged. Kraske's own statistics show how important this is. The summary of cases that he has published is small but very interesting. He operated on 14 cases of carcinoma of the pylorus and performed resection of the pylorus 4 times, gastro-enterostomy 10 times. Of these 14 cases, 6 died—that is, 43 per cent. These operations, however, were performed at two different periods in this author's experience: 6 of the operations were performed in the first period, 8 in the second; of the 6 that were operated in the first period, all died; of the 8 that were operated in the second period, none died. On account of the unfavorable results that Kraske obtained in the first period, he refused for a long time to operate on cases that were sent to him.

These statistics show that the operation *per se* is not dangerous, but that the unfavorable results obtained are chiefly due to the fact that the operation is attempted in cases where it is not indicated. Thus, many operations have been performed on patients who were very much reduced, and whose loss of strength had already been extreme.

Resection should only be performed if the tumor is still freely movable, if no metastases have formed, and if the patient possesses relatively good strength. If adhesions have formed (provided they cannot be easily separated), and if metastatic nodules are present in other organs, gastro-enterostomy should be performed if an operation is indicated at all.

Kraske believes that we shall gradually succeed in treating carcinoma of the pylorus by surgical means before this lesion can be palpated as a tumor, and even before it can be diagnosed with certainty. He claims that we should operate in every doubtful case in which a stenosis of the pylorus due to some organic tissue-change can be discovered. Even if a benign cicatricial stenosis is found, he advises operation. If, on the other hand, the stenosis is due to carcinoma, we can remove the neoplasm at a time when a radical operation offers good chances for recovery.

I certainly think that we can endorse the statement that an operation should be performed in all cases in which stenosis of the pylorus due to organic tissue-changes can be discovered. The character of the operation will, of course, depend on the nature of the stenosis. But let me ask, How do we make the diagnosis of stenosis of the pylorus in cases in which no tumor can be palpated? Our only criteria are motor insufficiency and ectasy; but wherever motor insufficiency and ectasy are marked, a tumor can usually be felt; in other words, the carcinoma, as a rule, is in an advanced stage. In many instances the tumor can even be felt before motor insufficiency and ectasy develop.

I have already mentioned a theory that Israel propounded in regard to the method of growth and the origin of carcinomata of the pyloric region. He observed that in cases of carcinoma of the stomach that were not situated directly at the orifices of the organ, but at some little distance from them, a narrow zone of healthy mucous membrane can usually be found in a portion of the pyloric region. This led him to assume that carcinomata do not start from the orifices of the stomach, but are merely arrested in their development when they reach them. If this is the case (and clinical experience seems to favor this view), we may expect to find that all carcinomata that produce symptoms of stenosis are already in an advanced stage. We certainly succeed in many cases in diagnosing carcinomata with a great degree of probability even when no symptoms of stenosis are present. If the general strength of the patient is good, an exploratory laparotomy may be performed in such cases. This operation, of course, is only indicated in those cases in which the presence of a carcinoma or of an organic stenosis of the pylorus can be determined.

The main sphere of operative interference will always remain carcinoma of the pylorus proper. Attempts have been made, it is true, to remove carcinomata that were situated in other portions of the organ, even if they were very large, by operative procedures. Resection of the pylorus must be considered the more radical method as compared to gastro-enterostomy, and should, in general, be preferred to the latter. Gastro-enterostomy only relieves the stasis of ingesta in the stomach and aids their passage into the intestine; in other words, it relieves motor insufficiency. Quite a number of cases are on record in which the patients enjoyed good health for years after resection. Kocher has reported the case of a woman who was in good health five and a half years after the operation. Wölfler also reports a case in which the patient was well for five years; at the expiration of this time a glandular metastasis recurred. If resection is impossible, gastro-enterostomy may be performed in order to relieve the symptoms of stenosis. The chief advantages of this operation are that it is simpler and more easy to perform; on the other hand, its effects are only symptomatic, for it merely relieves stagnation; but it may prolong life even though it exercises no influence on the development of the carcinoma. Gastro-enterostomy will always remain a late operation,—an operation directed against stagnation and against atony and its results. All these conditions, however, only develop after the cancer has existed for a long time. Resection of the pylorus should be performed early, and the sooner it is performed the better the prognosis. The more skill we acquire in diagnosis, the more the methods of diagnosis become improved; the sooner, therefore, we succeed in diagnosing carcinoma, the sooner will we be able to cure cancer of the stomach by a radical operation.

At the present time gastro-enterostomy ranks above pylorectomy. Let us hope that the art diagnosis will advance with such rapid strides that we will soon be able to recognize carcinomata very early in their development and at a time when a radical operation is possible. For the present, gastro-enterostomy is more popular because we recognize the disease so late, and willingly adopt any method that will even palliate the patient's distress.

Whether or not gastroscopy will become so perfected that this method of examination can be generally performed without great difficulty and without endangering the patient's life cannot be predicted. Rosenheim has recently done some very excellent work in this direction, and has contributed much to our knowledge of this method. For the present, however, it is too complicated and even too dangerous under certain circumstances to merit general adoption. It is not impossible that apparatus and appliances will be invented that are more easy to manipulate, and that some day we shall be able to make the diagnosis of carcinoma by means of their use at a much earlier stage than we can to-day. The method in vogue at present urgently calls for improvement.

This is not the place to enter into a discussion of the technique of the operations we have discussed. The surgeon is concerned with perfecting the technique of his methods. The internist should direct all his energies to improving the methods of diagnosis, so that the disease can be recognized early in its development.

TOTAL EXTIRPATION OF THE STOMACH.

Schlatter[1] reported the first successful total extirpation of the stomach in 1897, the patient surviving for a considerable period. It appeared that digestion was successfully accomplished by the intestines, and that the patient's suffering was relieved. Following this many operations of the kind were performed both here and abroad; the first in this country being done by Bernays, of St. Louis. Among the successful operations the results were similar to those reported by Schlatter. Owing to the great mortality and the early deaths that succeeded in those cases that were reported as successful, this procedure has for the most part been abandoned. It has been proved, however, that patients may survive for a period after extirpation of the stomach, with a fairly good digestion, and without diarrhea. Nevertheless, it may be said that the operation is now generally discredited.

OTHER TUMORS OF THE STOMACH.

As compared to carcinoma, all other tumors of the stomach are of subordinate clinical importance: for, in the first place, non-carcinomatous

[1] *Correspondenzbl. f. Schweiz. Aertze,* December 1, 1897.

neoplasms are very rarely found in the stomach ; in the second place, it is almost impossible to differentiate them from carcinoma during life. In addition to the atypic proliferation of gland tissue that we have spoken of above, and that may lead to the formation of polypous excrescences, sarcomata, lipomata, fibromata, and myomata have been seen in the stomach. Of these forms of tumor, the most important and relatively the most frequent are sarcomata.

Sarcomata of the stomach may be primary or secondary. The latter, with the exception of lymphosarcomata, are less frequently encountered than the former. Primary sarcomata of the stomach-wall, in particular myosarcomata and fibrosarcomata, as a rule present the appearance of circumscribed tumor nodules. Lymphosarcomata, on the other hand, appear as flat, extended infiltrations. The former, it appears, may develop from any point of the stomach, most frequently, according to Schlesinger[1] (who has studied sarcoma of the stomach very carefully), from the greater curvature.

These tumors may vary greatly in size and form ; they may be either spherical or of an irregular, nodular form ; may be attached by a broad base or by a narrow base. Occasionally they grow very large. Brodowski,[2] for instance, described a sarcoma of the stomach that weighed twelve pounds.

A variety of different sarcomata have been observed, as myosarcoma, fibrosarcoma, spindle-celled sarcoma, angiosarcoma. Sarcoma, like carcinoma, frequently leads to the formation of metastases in neighboring organs, particularly in the lymph-glands.

According to Schlesinger's summary, primary lymphosarcomata may occur at any age ; they appear most frequently, however, between twenty and thirty-five years. The other sarcomata seem to appear with greater frequency in older subjects. No marked differences can be discovered in regard to the frequency with which this form of tumor appears in the different sexes. Its etiology is wholly obscure.

The disease-picture, according to Schlesinger, is the following : The onset of the disease is usually insidious; all gastric symptoms may be absent, particularly in secondary sarcomata, or they may appear at a late stage of the disease. In many instances progressive emaciation is the first symptom presented ; in other cases, again, the disease begins with stomach-symptoms,—loss of appetite, a feeling of pressure and fulness in the region of the stomach, acid belching, and a disagreeable taste in the mouth. In other instances frequent attacks of vomiting and violent spasms of pain in the region of the stomach occur early in the course of the disease. All of these symptoms gradually become exacerbated, the loss of appetite increases, the attacks of vomiting become more frequent, and finally coffee-ground material may even be raised. If the tumor of the stomach can be palpated, it presents essentially the same peculiarities as carcinomatous tumors of the stomach. If the growth is situated at the pylorus, secondary dilatation of the stomach followed by stagnation of stomach-contents may result. In lymphosarcomata of the stomach dilatation of the stomach without stenosis of the pylorus may also occur.

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[1] *Zeitschr. f. klin. Med.*, vol. xxxii., supplement.

[2] *Virchow's Archiv*, vol. lxvii.

Tetany has been observed in this disease as in other stomach-lesions that are accompanied by dilatation of the organ (case of Fleiner[1]).

Investigations into the chemism of the stomach in this disease have failed to reveal any appreciable difference between sarcoma and carcinoma. Schlesinger has reported three observations that are very interesting in this respect. In the first case (myosarcoma) free hydrochloric acid was absent, and there was an abundant quantity of lactic acid. In the second case (lymphosarcoma) a gradual transition occurred from normal gastric function to a perversion of the function that was analogous to that seen in carcinoma,—that is, free hydrochloric acid was finally found absent, and lactic acid appeared in considerable quantities. This transition occupied considerable time. In both of these cases were found large numbers of the long bacilli that Schlesinger and Kaufmann consider characteristic for lactic acid fermentation. In the third case neither free hydrochloric acid nor lactic acid was found. In all three cases blood was repeatedly discovered in the stomach-contents.

From a clinical point of view, therefore, we cannot say that essential differences exist between the chemism of the stomach in carcinoma and sarcoma; nor are there any other very pronounced differences. Hematemesis occurs in both diseases. No essential differences in regard to the appearance and the course of the cachexia exist in the two diseases. The duration of the disease fluctuates from several months to three years, the average duration being from one to one and a half years.

It will be seen, therefore, that neither the local nor the general symptoms furnish any positive information that might enable us to make a differential diagnosis between carcinoma and sarcoma.

Schlesinger, however, has shown that a number of factors may be utilized in the differential diagnosis. The location of the metastases and the changes in the spleen are both important in this respect. Primary sarcoma of the stomach forms metastases in the skin with relative frequency. These secondary deposits assume the form of small or large nodules. This fact frequently enables us to make the diagnosis if these cutaneous nodules can be excised without difficulty and subjected to histologic examination. Occasionally particles of tumor may also be vomited or be discovered in the stomach-contents; these, too, of course, can be utilized for the diagnosis. Observations of this kind, however, are not recorded in the literature so far. A few years ago I observed a case of this character in which a large sarcoma nodule was vomited during an attack of hematemesis. In this case there were also numerous metastases in the skin. Schlesinger attaches particular importance to the metastases in the intestine in diagnosing lymphosarcoma. According to Kundrat,[2] metastatic lymphosarcomata of the intestine never produce strictures, but usually lead to dilatation of the intestine.

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[1] *Arch. f. Verdauungskrankh.*, vol. i.

[2] *Wien. klin. Wochenschr.*, 1893, No. 12.

If, therefore, we find tumors of the intestine without the signs of stenosis, the diagnosis of lymphosarcoma is probable (according to Schlesinger). The greater the swelling of lymph-glands, the more probable the diagnosis of lymphosarcoma. Schlesinger calls attention to the fact that carcinomatous tumors of the intestine always produce symptoms of stenosis.

According to this same author, the swelling of the spleen can be utilized in the diagnosis. In a relatively large number of cases, both of primary and secondary sarcoma and lymphosarcoma of the stomach, the spleen is swollen. Swelling of the spleen was observed in all the cases that Schlesinger examined; and as it is very rare in carcinoma of the stomach, this factor might possibly be utilized in rendering a differential diagnosis.

An examination of the buccal cavity may also furnish some information. According to Kundrat, the tongue is changed in a characteristic way. The follicles are swollen and tumefied. The symmetric arrangement of the nodules, ridges, and papillæ is particularly conspicuous; they extend in rows from the median line of the base of the tongue to both sides and forward. Secondary lymphosarcomata always form broad infiltrations on the tongue.

Notwithstanding all these points, a positive diagnosis can rarely be made. This is well illustrated by a case that Leube[1] reported, in which there were universal sarcomatosis of the skin and a genuine epithelial carcinoma in the stomach.

[Dock[2] reports a case of primary sarcoma of the stomach in a man fifty-five years old. The tumor was removed by Nancrede, and the patient reported well several months after the operation. The successful removal of a sarcoma from the stomach of a man is reported by Morton.[3] In reviewing the recent literature on the subject Dock found that several forms of primary sarcomatous growths occur in the stomach, and that, as a secondary growth, they occur in the stomach perhaps more frequently than secondary cancer. The onset of the disease is apt to be insidious, as in the case reported by Baldy,[4] in which the disease had never distressed the patient, and was attended with unusually good appetite. Finlayson reports an instance of the spindle-celled variety of sarcoma occurring in the stomach of an infant three years old. The results of physical examination closely resemble those found in carcinoma, and in the few cases in which examination of the stomach-contents has been made the findings closely approached those commonly discovered in the stomach-contents of carcinoma.

According to Brooks,[5] who reports a case of multiple sarcoma of the stomach, the growth usually appears at the lesser curvature of the stomach, but it may appear in the greater curvature or at any other point.—Ed.]

The treatment should be based on the same principles as the treatment of carcinoma. In lymphosarcomatosis, however, the administration of arsenic may be attempted.

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[1] *Specielle Diagnose d. inneren Krankh.*, third edition.

[2] *Trans. Assoc. of Amer. Phys.*, May 3, 1900.

[3] *Brit. Med. Jour.*, June 1, 1901.

[4] *Jour. Amer. Med. Assoc.*, 1898, vol. i., p. 523.

[5] *Med. News*, May 14, 1898.

[**Apparent Tumors of the Stomach.**—The presence of a tumor in the region of the stomach, transient in character and apparently depending upon spasm, is occasionally noted. Such was the case reported by Schnitzler,[1] who, after a laparotomy, was enabled to see the stomach contract, forming a tumor the size of a small apple. It was supposed that there occurred a spasm in the vicinity of a round ulcer. The case was treated by pyloroplasty. Such spasmodic tumors are not often demonstrated in this way, although they probably occur oftener than is supposed. Apparent tumors of the abdomen, more or less continuous in their manifestations, are occasionally seen, and sometimes offer no little embarrassment in diagnosis. Einhorn[2] collects 23 cases in his own practice, most of which were associated with either gastroptosis or enteroptosis. This author supposes the apparent tumor to depend upon prolapse of the left lobe of the liver, exposure and thickening of the abdominal aorta, hypertrophy of part of the abdominal muscles, and possibly adhesions around the lesser curvature of the stomach. In gastroptosis the pancreas may be palpated, especially when it is in a state of induration, and this has been mistaken by some for gastric tumor.—Ed.]

FOREIGN BODIES IN THE STOMACH.

Quite frequently foreign bodies enter the stomach and produce more or less violent distress. Foreign bodies are swallowed either on purpose or unintentionally, or foreign material may gradually be deposited in the stomach. The clinical symptom-complex will vary according to the character of the foreign body present in the stomach. Many foreign bodies, particularly if they are small and have a smooth surface, are removed from the stomach by vomiting after a short or a long time. Others succeed in passing through the stomach and intestine, and are finally deposited with the stools. In still other cases the foreign body produces inflammatory symptoms in the intestine that may lead to invagination, to the formation of abscesses, or to perforation. In this section we will discuss only those cases in which the foreign body remains in the stomach.

A great variety of foreign bodies have been found in the stomach—for instance, forks, knives, spoons, artificial teeth, balls of hair, needles, pieces of iron, nails, pieces of lead and of wood, stomach-pumps, and other articles.

Friedländer[3] and Vonnegut[4] have each described a case in which shellac stones led to the formation of a tumor in the stomach. These patients had been working with an alcoholic solution of shellac, and for a long time had swallowed small quantities, and this had ultimately led to the formation of these stones in the stomach.

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[1] *Centralbl. f. Chir.*, September 3, 1898. [2] *Med. Record*, November 24, 1900.

[3] *Berlin. klin. Wochenschr.*, 1881, No. 13.

[4] *Deutsch. med. Wochenschr.*, 1897, No. 26.

Schreiber[1] describes a case of freely movable tumor of the stomach that was removed by operation, and was found to be a mass of vegetable fibers. Richter[2] reports a case in which a tumor formed of sarcina ventriculi occluded the pylorus. Leube[3] reports the case of a woman who allowed the stomach-sound to drop into the stomach during lavage. In this instance the tube remained there for nine days, and was finally evacuated by vomiting.

Only those cases are clinically interesting in which the foreign body remains in the stomach. The local disturbances may be very severe or may be altogether absent. So long as the foreign body is not deposited with the stools or vomited we are confronted with the task of removing it artificially.

The diagnosis of foreign bodies in the stomach is self-evident in the majority of cases. As a rule, the history will inform us that the patient, for instance, swallowed the stomach-tube while performing lavage of the stomach or swallowed some foreign body, either intentionally or by accident.

The diagnosis, of course, is more difficult in those cases in which a tumor is felt in the region of the stomach, but in which the anamnesis gives us no information in regard to the entrance of a foreign body into the stomach. This is illustrated, for instance, by the cases of shellac stones that were described above.

The discovery of a tumor in the stomach is the most important point for the diagnosis. If the tumor is movable, this indicates that the swelling may be a foreign body; the tumor, however, must possess more than the slight degree of motility that we occasionally feel in other tumors that are adherent to the stomach-wall. It must be very movable, so that the tumor can be pushed about in every direction, and always occupies the lowest point of the stomach as soon as the patient changes his position. These factors alone determine the presence of a freely movable tumor in the stomach. The nature of the tumor, of course, is not revealed by this discovery. In some instances we may succeed in feeling the foreign body with the sound; in other instances the electric magnet may be employed with some effect. Chemical examination of the stomach-contents can hardly give us much information. In some cases transillumination with Röntgen rays may be employed. The chief indication for treatment is to remove the foreign body from the stomach.

We should never attempt to remove the foreign body by the same way in which it entered the stomach, unless we can determine that it is small, smooth, and possesses no sharp angles. If this is the case, some emetic, preferably apomorphin, may be administered. I repeat expressly that this method of treatment is justified only if we know the character of the foreign body. If the foreign body is small and smooth, however, it usually passes into the intestine and leaves the body by the natural passages without further detriment to the patient.

Richter treated the case in which the pylorus was occluded by sarcina (see above), and in which aspiration of the stomach-contents revealed nothing but sarcina, by washing the stomach alternately with solutions of hyposulphite of sodium and corrosive sublimate. In this way he arrested the development of the parasite and succeeded in bringing about marked improvement.

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[1] *Deutsch. med. Wochenschr.*, supp., 1897, No. 4. [2] *Virchow's Arch.*, 1887, vol. cvii. [3] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii.

If the foreign body is large, the only way to remove it is by an operation. In the majority of cases surgical interference has been postponed until very late—that is, six months and even years after the foreign body entered the stomach. Operative removal of foreign bodies has only rarely been performed early—that is, within the first three or four days. Expectant treatment is certainly not to be recommended. [When sharp objects find their way into the stomach and are in danger of producing serious trauma, it is recommended by Johnston[1] that the patient be fed sandwiches that contain cotton-wool, the fiber of which is expected to wrap itself about the foreign body and thus enable it to pass the pylorus without doing injury. In Johnston's case, in which a man swallowed a metallic plate of teeth, the passage of the article was safely accomplished.—Ed.]

The presence of small metallic bodies having sharp points or cutting edges is a source of danger, and their removal has been a matter of great difficulty even when laparotomy is resorted to. An ingenious method for the extrication of such bodies is described by S. Mayou.[2] His device consists of a cylindric electro-magnet, 2 inches long and $\frac{5}{16}$ of an inch in diameter, with a soft iron core exposed at the ends, and having a lifting power of four ounces when connected with an ordinary four-volt battery. This magnet, connected with properly insulated wires, is inserted through an ordinary stomach-tube, open at the distal extremity. This stomach-tube has a narrow silver band at its end that permits one to know its exact position in the stomach by means of the X-ray apparatus. The tube having been introduced, the magnet is passed to its distal end. The patient then lies upon a couch that permits the application of the X-rays beneath the patient. By suitable manipulations and guided by the X-ray shadows, the end of the stomach-tube is brought near the foreign body. The current is then turned on and the foreign body is drawn into the tube, after which the tube, including the magnet and the foreign body, so covered that it cannot injure the mucosa, is slowly withdrawn. This ingenious device ought to be successful in cases to which it is adapted.

Fricker[3] has published some statistics on gastrotomy for foreign bodies in the stomach, and I learn from them that this operation has so far been performed 53 times. This figure is probably too small, as some cases of this kind may not have been published. Fricker himself reports a case in which he removed 37 foreign bodies from the stomach—namely, one key, two teaspoons, one fork, two wire nails, two hairpins, twelve pieces of glass, one window-hook, one steel pen, nine sewing-needles, one piece of graphite, one shoe-button, one grape-kernel, two balls of tin-foil, and one crochet-needle. Marcet[1] reports the case of a sailor who was accustomed to performing the trick of swallowing knives. On autopsy, 30 knife-blades were found in the stomach. In the majority of cases, however, a single foreign body has been found.

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[1] *Brit. Med. Jour.*, March 29, 1902.

[2] *London Lancet*, Dec. 6, 1902.

[3] *Deutsch. med. Wochenschr.*, 1896, No. 4.

If the presence of a foreign body in the stomach can be established, an operation should be performed at once, for there is always danger of serious injury or perforation of the stomach-wall. As a matter of fact, a number of cases are on record in which perforation followed soon after the entrance of the foreign body into the stomach. In other cases again ulcerations formed, the stomach became adherent to neighboring organs, or other complications developed.

The results of the operative treatment of this condition are generally favorable. Recovery ensued in 81.4 per cent. of all cases, and many of these operations were performed late. The results of an operation are apt to be more favorable the sooner gastrotomy is performed.

[**Syphilis of the Stomach.**—As to the frequency of syphilitic affection of the stomach, some difference of opinion exists among observers. However, a considerable number of cases demonstrated by autopsy to be syphilitic disease are now recorded, and it may be said that gastric syphilis rests on a safe pathologic basis. Soltau Fenwick[2] states that syphilis may affect the stomach in three ways: by the formation of gummata, by producing endarteritis, and by exciting chronic inflammation of the mucosa. He holds that the chief point in differential diagnosis is the intractability of the case to ordinary methods of treatment, while the symptoms subside rapidly upon the administration of antisyphilitic remedies. Flexner[3] expresses the belief that syphilitic gastric ulcer is not rare, and cites a case in which he concluded that the ulcer was brought about by the combined softening of the submucous gummatous infiltration, and by obstruction and obliteration of blood-vessels in the same situation. Einhorn[4] reviews the subject and expresses the opinion that syphilis of the stomach is by no means of rare occurrence. He classifies its manifestations in the following order: (1) gastric ulcer of syphilitic origin; (2) syphilitic tumor of the stomach; (3) syphilitic stenosis of the pylorus—and recites a number of illustrative cases. He also, in a bibliography, collects the history of a number of cases reported by modern observers, most of whom refer to gastric syphilis as a rather unusual event.

According to Dieulafoy,[5] when signs of simple gastric ulcer appear in a syphilitic, it may be presumed that the lesion itself is specific. These lesions may appear under various forms, such as hemorrhagic erosions, ecchymoses, gummata, infiltration of the submucosa, and circumscribed gummatous ulceration, and cicatrices of these. He calls attention to the symptoms—epigastric pain, vomiting, hematemesis, and melena.

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[1] *Med.-Chir. Trans.*, vol. xii. p. 72.

[2] *London Lancet*, September 28, 1901.

[3] *Amer. Jour. Med. Sci.*, October, 1898.

[4] *Phila. Med. Jour.*, February 3, 1900.

[5] *Gaz. heb. de méd.*, 1902.

In 1894 I reported 12 cases of gastric syphilis[1] in which the diagnosis was based on clinical evidence. These cases occurred among 500 consecutive patients suffering from various stomach diseases, and, for the most part, complained of sensory and motor disturbance, regurgitation of gas, nausea, distress after eating, delayed motion, and gastralgia. In only 1 of the 12 was benefit seen to follow the ordinary measures of treatment, but they responded promptly when antisyphilitic medication was adopted, and were apparently cured. Since that time a good many cases have come under my observation, and I am led to believe, with Einhorn, that syphilis of the stomach is not rare. Allen Jones has reported a series of cases of gastralgia that depended upon syphilis. A review of those cases that have come to autopsy leads one to conclude that the lesions most common are gumma and increased cicatricial tissue. Undoubtedly instances of syphilitic arteritis of the stomach, as mentioned by Fenwick, are seen. Originally I was inclined to attribute the symptoms found in these cases to functional disturbances the result of autointoxication, but with further experience it seems probable that in most instances there is a distinct lesion of the stomach. The sudden improvement for the better under the influence of mercury or potassium iodid in a case that formerly was showing no improvement, and that was accompanied by manifestations of serious disease, has been found the most reliable criterion on which to base a diagnosis. Gastric syphilis is almost uniformly improved or cured by treatment; but when the disease is far advanced and accompanied by notable involvement of other viscera, like the case reported by Cæsaris-Demel,[2] cure is hardly to be expected. In not a few of the reported cases death has resulted from perforation occurring through a broken-down gumma.—ED.]

NERVOUS AFFECTIONS OF THE STOMACH.

LITERATURE.

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[1] *Med. News*, December, 15, 1894.

[2] *Brit. Med. Jour.*, January 27, 1900.

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Introductory Remarks.—The history of neuroses of the stomach may be divided into different periods. In the publications of the seventeenth and eighteenth centuries we frequently read of dyspepsia that is not due to any material changes in the stomach. In the first half of the last century, at a time when pathologic-anatomic methods of research were being introduced, the field of purely nervous disorders of the stomach became more and more circumscribed. Finally the attempt was made to deny the existence of purely nervous disorders, and to explain all the clinical pictures presented by some pathologic-anatomic lesion. In this way it happened that nervous diseases of the stomach disappeared for some time altogether from the pathology of diseases of the stomach. Gradually, however, they regained their place, for in many diseases of the stomach no anatomic basis could be discovered, so that clinicians and pathologists were forced to recognize the existence of purely nervous and functional disorders of the stomach.

The first valuable contributions to this subject were made in the early forties. I must confess that even to-day the question cannot be regarded as settled. We are not as yet basing on a solid fundament when we speak of purely nervous and purely functional diseases of the stomach. In Germany it was Leube, above all, who created a place for nervous dyspepsia in the general pathology of diseases of the stomach. As early as 1878 Leube, in his dissertation on "Diseases of the Stomach," in von Ziemssen's *Hand-Book*, arranged a scheme of the different diseases of the stomach that could be attributed to perversions of the nervous function of the organ. This scheme remains a model of thoroughness to-day. Leube subdivided the nervous diseases of the stomach into three groups:

(*a*) Cases in which the sensibility of the organ is increased or reduced.

(*b*) Cases in which the contractility of the organ is increased or reduced.

(*c*) Cases in which the secretion of the organ is increased or reduced.

It is true that this scheme was purely theoretic, for clinical disease-pictures that correspond to these different hypothetic perversions had not been described at that time. For this reason, too, Leube furnishes a good description only of gastralgia, and limits himself to a description of those forms of nervous dyspepsia alone in which the digestion was normal in regard to time and chemism, but in which certain dyspeptic symptoms appeared nevertheless.

In a lecture delivered to the Third Congress for International Medicine in 1884 Leube expressed himself in regard to nervous dyspepsia as follows: "Only those disease-pictures are to be included under the heading of nervous dyspepsia in which the digestion is normal both in regard to time and to chemism, but in which, nevertheless, the symptom-complex of dyspepsia is produced exclusively by some perversion of the nervous system, particularly of the nerves of the stomach; in which, finally, no objection can be formulated from an anatomic point of view against regarding the disorder of the nervous system as the primary basis of the dyspeptic symptoms."

According to Leube, one of the characteristic features of this form of dyspepsia is that the patients, during the time of digestion, develop certain symptoms that point to direct involvement of the nervous system. Such symptoms might be headache, vertigo, lassitude, belching of tasteless and odorless gas, frequent nausea, even vomiting, a feeling of fulness and pressure in the epigastrium that may occasionally develop into true pain, heartburn, a feeling of globus, varying appetite, and depression. All these symptoms are the most important ones to be observed. They may all appear together, or only a few may be presented. Leube himself, however, emphasizes particularly the fact that the diagnosis of nervous dyspepsia can never be more than a probable diagnosis, even though this whole symptom-complex be completely developed.

This symptom-complex, then, according to Leube himself, cannot be regarded as an infallible proof of the existence of nervous dyspepsia, for it is observed in many cases of organic disease of the stomach. It is important, therefore, in rendering the diagnosis, to determine whether other nervous or neurasthenic symptoms complicate or parallel the dyspeptic symptoms that are supposed to be due to some nervous disorder. We undoubtedly encounter cases in which the symptoms of nervous dyspepsia are due to some purely local trouble, but in many—probably the majority—of the cases the abnormal reactions of the nerves of the stomach are merely one symptom of general neurasthenia or hysteria. Hysteria, as we know, may become manifest in certain organs,—for instance, in the vocal cords, producing hysteric aphonia; but in most of these cases we can usually determine from a number of other symptoms that the lesion is of central origin—is hysteric; in the same way we may be able to determine that the dyspeptic symptoms are of central origin if we discover other symptoms of general nervousness, neurasthenia, or hysteria.

But even the existence of other nervous symptoms does not demonstrate absolutely that the local symptoms in the stomach are due to an abnormal reaction on the part of the gastric nerves, for purely nervous or neurasthenic perversions of the stomach-function may occur together with anatomic lesion of the organ. Both may be altogether independent of each other, or divers nervous disturbances may secondarily be caused by the primary disease of the stomach.

Leube threw a great deal of light on this subject when he introduced the analysis of the chemism of the stomach into the differential diagnosis of these complicated conditions. He argued that if the time of digestion was found to be normal and the chemism of the stomach was also normal, while at the same time, the patient complained of intense dyspeptic disturbances, this disproportion between the objective findings and the subjective symptoms indicated that the dyspepsia was nervous in character. If the stomach was found empty seven hours after a test-meal, Leube concluded that digestion was normal. Nowadays this finding is no positive criterion, for it demonstrates merely that the motor powers of the stomach are normal, but tells us nothing in regard to its secretory powers. We have shown that the stomach may get rid of its ingesta within a normal time, whether the secretion of gastric juice is abnormally increased or abnormally decreased, provided only the motor functions of the stomach remain intact.

All that Leube's method, therefore, shows is that the motor power of the stomach is not reduced; nevertheless, the motor powers of the stomach may be abnormal in another direction—namely, may be increased. It is true that a healthy stomach gets rid of a test-meal in seven hours at the latest, so that at this time the stomach is found empty; but we are not justified in inverting this proposition by saying that the stomach must be healthy or cannot be afflicted with any anatomic lesion if it is found empty at the expiration of seven hours.

I do not by any means wish to belittle the significance of aspiration of the stomach-contents in the diagnosis of nervous dyspepsia; on the contrary, it seems to me this procedure is absolutely indispensable if we wish to establish the nervous character of the disease. Nowadays, however, we should not limit ourselves to determining that the stomach is empty seven hours after a test-meal; we should endeavor to procure some of the stomach-contents at the height of digestion, study the quantity, the appearance, the peptic power, the acidity, the abnormal constituents, etc., of the material aspirated. This is a general rule that applies not only to cases of suspected nervous dyspepsia, but to all other diseases of the stomach. We should never diagnose the form of dyspepsia that Leube was the first to describe unless we study the gastric digestion with the aid of all modern adjuvants that we possess for testing the secretory and motor powers of the organ. Only if digestion is found to be normal by these means may we make this diagnosis.

Leube himself expressed the opinion, many years ago, that there are several forms of nervous dyspepsia, and that the one we have outlined, in which there are symptoms of sensory irritation and in which the patient complains of abnormal sensations at the time of digestion, is not the only form, though by all means the most frequent one. We know, nowadays, that there are not only sensory neuroses of the stomach, but also nervous disturbances of the secretory and motor functions of the organ.

The diagnosis of these purely sensory neuroses may be quite difficult. I need only mention the symptom-complex that appears when the stomach is adherent to neighboring organs. Here the patients may complain of symptoms that are altogether analogous to those that we have described above, and, at the same time, an analysis of gastric secretion and motility will reveal normal conditions. It is well known that cases of this character frequently present great diagnostic difficulties; if the patient, in addition, presents a variety of nervous and neurasthenic symptoms, or, finally, lapses into a state of hypochondriasis, the difficulties are enhanced. We see the development of these general functional neuroses quite frequently, particularly as the patients are apt to become hypochondriacal and neurasthenic after consulting many physicians in vain and looking for aid and relief in many different places. Every physician has probably seen cases that were diagnosed as nervous dyspepsia for a long time, until finally some material cause for their symptoms was discovered.

I quote this merely as one of the many difficulties that are encountered in rendering the diagnosis of nervous dyspepsia. Even if the patients are examined with the greatest care, errors of this kind may occur.

As a rule, gastric neuroses are defined as functional disorders that are not based on any pathologic changes of the gastric wall. If we could inspect the interior of the stomach at any time, or if the clinical symptoms that correspond to definite anatomic changes were so typical that the former could be diagnosed from the latter, this definition would be sufficient; but, unfortunately, this is not the case. Even if we can determine with certainty that the symptom-complex presented does not correspond to any of the anatomic lesions of the stomach that we are able to recognize with the means at our disposal to-day, this does not enable us to make the diagnosis of nervous dyspepsia. For the present, it is true, we may be justified in considering the disturbances as functional, but we do not know whether they are nervous or not. We know a great many diseases that present a well-rounded clinical disease-picture, the anatomic basis of which has never been discovered. The nature of these diseases and their primary seat are not clear; at the same time, we assume that they are due to some definite lesion that has not yet been discovered.

I think that Oser[1] is correct when he says, "Clinicians and practising physicians cannot wait for the discovery of an anatomic basis for each disease, but must construct different types of disease as soon as they are recognizable." This, however, should not justify him in declaring a disease to be a neurosis whenever the anatomic basis is unknown. This caution, it appears to me, applies with greater force to the stomach than to any other organ, for here the anatomic examination is particularly difficult. In order to consider a disease a neurosis we should be able to determine that the disease is a disturbance of innervation, and we should be able to state with certainty that no objections to this view can be formulated on anatomic or clinical grounds.

I do not doubt that many diseases that we consider nervous to-day will in time be found to be based on pathologic-anatomic changes. For the present, however, I do not think we are justified in declaring any group of gastric symptoms that does not fit into the frame of the few anatomic disease-forms that we have discovered so far a neurosis or an anomaly due to perverted innervation of the stomach.

In order to declare a disease a neurosis we should possess not only negative clues, but also positive ones. We should speak of a nervous disorder only if we have direct evidence that the symptoms of the disease are nervous in character. For all these reasons I think that the etiology of these conditions should be studied with particular care.

Every physician who has had much experience will agree with me when I say that it is frequently very difficult, even with the aid of all our modern methods, to differentiate an organic disease of the stomach from a neurosis of the stomach. Nowadays we are inclined to take a view opposite to that of our immediate clinical predecessors. Formerly, as I have said, clinicians attempted to force all the disease-pictures into the narrow frame of the few syndromes that were known to be founded on a pathologic-anatomic basis. To-day, it appears to me, we are inclined to err in the opposite direction, and to declare every disease a neurosis that does not coincide with one of the established clinical entities. I need only recall the innumerable disease-pictures that were formerly included under the term of chronic catarrh of the stomach. Any disorder of the stomach that was not an ulcer, carcinoma, or an ectasy, was called a chronic catarrh. Nowadays, we demand a certain amount of positive evidence before the diagnosis chronic catarrh of the stomach or chronic gastritis is accepted. All negative evidence is considered insufficient.

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[1] *Die Neurosen des Magens und ihre Behandlung*, Vienna and Leipsic, 1885.

We see, therefore, that the section on Neuroses of the Stomach is incomplete, and that the conception of this disease is not grounded on a solid fundament. We are undoubtedly predisposed to nervous diseases in this day and generation, and it may be due to this that clinicians are more inclined than formerly to call diseases of the stomach that do not fit into the frame of typical and established symptom-complexes neuroses. This tendency, of course, is a prolific source of error.

Etiology.—In order that a neurosis of the stomach may develop there must be a particular predisposition to this disease. Certain nerve-tracts must either be overirritable or their irritability must be reduced. This abnormal irritability may be seen in the secretory, the motor, or the sensory areas of the stomach, and may involve one of the spheres, or several at once, or all together. In one series of cases the nervous apparatus of the stomach is the primary seat of the disease; in another, the gastric disturbances are due to reflex disorders that are due to disturbances in other organs; the latter are called reflex neuroses of the stomach.

Inversely, perversions of gastric function may reflexly involve other portions of the nervous system, even the higher psychic centers. The latter disorders have been called neuroses or psychoses of gastric origin.

This class of diseases cannot, of course, be included under the heading of neuroses of the stomach proper, for the nature of the disease is not a disturbance of gastric function, but an abnormal reaction on the psychical side—*i. e.* of the central organ. Even normally the act of digestion leads to a certain depression of the mental and bodily functions. Most people feel tired after a large meal, and are not inclined to mental or physical labor. We need not be surprised to find that a reaction of this kind that is seen even in healthy subjects may, under certain circumstances, be abnormally strong, particularly in subjects whose nervous system is predisposed to reflex irritation. In many of these subjects we see attacks of fear, asthmatic attacks, disturbances of the heart-beat, attacks of vertigo, etc.

Nervous symptoms of this kind that appear when the functions of the stomach are normal do not interest us in this place. We can discuss only those forms of nervous disorders that follow in the train of certain diseases of the stomach. In order that such nervous disorders may originate there must, of course, be a particular predisposition, otherwise we could not understand why reflex symptoms of this kind appear in one patient and not in the other, even though both subjects may be afflicted with the same stomach disease; we could not understand, moreover, why an attack of acute indigestion produces migraine in one subject, violent palpitation in another, angina pectoris or an attack of asthma in a third subject. The particular predisposition determines the direction of the reflex irritation. In one person the irritation is transmitted to the pulmonary fibers of the vagus, in another to the cardiac, in a third to the sympathetic fibers. Even the higher psychic centers or the motor area may be reflexly irritated in certain diseases of the stomach. I will merely call attention to the fact that in atrophy of the gastric mucosa accompanied by severe forms of anemia true pareses are occasionally observed. The atrophy of the stomach can certainly not be considered the cause of these paralyses of peripheral muscles. Singultus or chronic spasms of the diaphragm are more directly related to certain diseases of the stomach. The attacks of tetany that are seen in certain cases of dilatation of the stomach, particularly if this condition is complicated by the continuous secretion of gastric juice, are also neuroses—more specifically, motor neuroses—of gastric origin. I refer to the section on Dilatation of the Stomach and Continuous Secretion of Gastric Juice for the discussion of the last-named form of neurosis. In that place I mention it as a rare sequel of certain types of gastrectasy.

In the cases just enumerated we are not dealing with a neurosis of the stomach proper,—that is, with disturbances of the nerves of the stomach and of the stomach itself,—but with disturbances of other nervous areas; these are due to reflex irritation emanating from the diseased stomach. I will refrain from discussing these forms in detail, for they are not really true neuroses of the stomach. This is evidenced by the fact that the same nervous disturbances may be seen in diseases of other organs, provided the subject is predisposed to these reflex disorders. I have discussed many of these disturbances as far as I thought it necessary in treating of the sequelæ of certain diseases of the stomach.

Those disorders of the stomach that are due to psychic irritation are more interesting. Numerous neuroses of the stomach belong to this category. Even under ordinary circumstances psychic excitation and emotional disturbances may exercise an influence on the functions of the stomach. I need only recall the influence that psychic or emotional disturbances exercise on the appetite. Occasionally we see that violent psychic impressions influence the secretion of gastric juice and the peristaltic action of the stomach and intestine. As a rule, these disturbances of gastric function are transitory. In some cases, however, disturbances of this kind persist for some time and show a tendency to recur, particularly in hysteric subjects, in neurasthenics, and in persons who are exhausted by severe mental overexertion. In the latter class of patients, the nervous disorders of the stomach may become so conspicuous that the diagnosis of a genuine primary disease of the stomach may be made. The patients complain almost exclusively of stomach-symptoms, and the only way to avoid error is to elicit a careful history and conscientiously to weigh all the factors presented.

Secondary neuroses of the stomach are also occasionally seen in pathologic processes of the brain and spinal cord. One of the best-known examples of this kind are the gastric crises of tabes, but even in this disease the symptom-complex presented is not uniform in all cases. Sometimes we see violent attacks of gastralgia or, again, spasmodic seizures that occur simultaneously and may lead to vomiting, or, finally, disturbances of gastric secretion. In some of these cases of gastric crises large quantities of gastric juice are vomited containing much hydrochloric acid. Cases of this character are undoubtedly secretory neuroses of the stomach due to morbid irritation of the nerve-tracts leading to the stomach, an irritation that originates from the central nervous system. In certain diseases of the brain and the meninges purely functional disorders of the stomach, as nausea and vomiting, are also frequently seen.

Reflex disturbances of the stomach may originate from organs other than the central nervous organs. Certain diseases of the liver, the intestine, the kidneys, and the sexual organs may produce functional disturbances of the stomach. As a matter of fact, any painful irritant may, under certain circumstances, produce reflex perversions of the gastric functions. Violent pain in any portion of the body, as a rule, exercises a distinct influence on the appetite, and may even produce nausea and vomiting.

In attacks of gall-stone or kidney colic we almost always see gastric symptoms—nausea, belching, and vomiting. In some cases these symptoms are more conspicuous than the symptoms of the primary disease, so that a primary gastric affection may be simulated. As soon as the irritation of the bile-passages or of the kidney ceases the gastric disturbances that are due to reflex irritation also stop.

It is a well-known fact that reflex perversions of the gastric function may originate from the sexual organs. Even in healthy women menstruation frequently exercises a distinct effect on the functions of the stomach and leads to decided perversions of gastric secretion. In many women in whom menstruation itself produces no distress there may be loss of appetite, nausea, and other gastric discomfort during this time. Gastric disturbances of this character are particularly pronounced if the uterus or the adnexa are diseased.

Another condition that must be grouped with these reflex neuroses is pernicious vomiting of pregnancy. Most gynecologists attribute this symptom to centripetal irritation of the uterus, transmitted along the sympathetic fibers and causing reflex vomiting. Other investigators claim that hyperemesis during pregnancy is due to some morbid process going on within the uterus. Pernicious vomiting of pregnancy, however, is usually due to some morbid predisposition of the nervous system. We shall find that in the majority of cases reflex irritability is increased; this alone will explain those cases in which vomiting that persistently resisted all measures suddenly stops after some psychic or somatic influence. Kaltenbach,[1] for instance, reports a case in which a woman vomited so persistently that she was emaciated to a skeleton. As soon as the physician informed her that artificial abortion, which she dreaded very much, would positively be induced the next day, the vomiting suddenly stopped, never to recur.

Cazeaux reports the case of a young woman who was suffering from persistent vomiting that could not be stopped. Her husband suddenly developed invagination of the bowel and was declared in danger of his life. The excitement incident to this event stopped the vomiting at once. I think that Kaltenbach is correct in his view that pernicious vomiting of pregnancy is similar to hysteric vomiting. This author reports another interesting case of a young woman who began to suffer from obstinate vomiting very soon after conception. The condition became so aggravated that she could retain nothing whatever in the stomach. The patient was given the suggestion that harmful masses were present in the stomach that had to be removed, and that as soon as they could be gotten rid of vomiting would stop at once. The stomach was washed out and the patient was informed that the vomiting would stop; as a matter of fact, she did not vomit any more after that.

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[1] *Zeitschr. f. Geburtsh. und Gynäkologie*, vol. xxi., No. 1.

Whether or not Kaltenbach's view that the vomiting of pregnancy is due to hysteria applies to all cases remains to be determined. It is certainly well to remember that the hyperemesis of pregnant women may under certain circumstances be one of the manifestations of a central neurosis—of hysteria.

[As earlier stated, eye-strain has been found a frequent cause of functional stomach-trouble, especially among those who are living an indoor life and are accustomed to the close use of the eyes. This is particularly true in cases of mixed astigmatism and anisometropia, and is often associated with muscular imbalance.—Ed.]

Gastric symptoms are occasionally seen as the result of autointoxication—for instance, in uremia. It is well known that vomiting constitutes one of the early symptoms of uremic intoxication.

The following predisposing and causative factors of neuroses of the stomach may be mentioned. The condition is more frequent in women than in men. This is due to the fact that women are usually more high-strung than men, and that a number of factors, like menstruation, diseases of the sexual organs, pregnancy, etc., affect the female sex alone. Neuroses of the stomach are, however, quite frequent in men, and are usually due to a variety of agencies that cause overirritation of the nervous system.

The age of the patient is of some importance, not only for the genesis of neuroses of the stomach, but for neuroses in general. We can readily understand why these states develop particularly during the years in which the nervous system is most severely taxed. Nervous disorders of the stomach are usually encountered earlier in women than in men. This is due chiefly to the fact that women and girls are exposed to many influences that damage the nervous system at an earlier period of their life than of men. In advanced years neuroses of the stomach are rare. If they are found in old people, they usually date from some former time.

Heredity, too, plays an important rôle. We quite frequently find that a tendency to nervousness or hysteria is transmitted from one generation to another. This applies particularly to women.

The external circumstances under which the patient lives are also of great importance. In this respect a very considerable difference can be observed between the two sexes; in men, neuroses of the stomach are more frequent in the higher and better educated classes, whereas in women no great difference between the different classes is noticed. In men we see neuroses of the stomach particularly at an age when the greatest demands are made on the mental powers—that is, in the middle years of life. The disease appears particularly in men whose occupation is exciting and leads to an irregular mode of life. In married women and young girls I do not find the difference between the better and the lower classes so marked, for while it is true that nervous disorders of the stomach are more frequent in the higher and more effeminate classes, we encounter the disease very frequently in the lower classes—for instance, in servant-girls and in peasant women. This need not surprise us, for we have seen that the female sex is more predisposed to neuroses in general than the male. Finally, we need not be surprised to find exhaustion and hyperirritability of the nervous system in the men of our day who are occupied in a strenuous struggle for existence, for recognition, and success, and who, on the other hand, indulge in many abuses that reduce their powers of resistance. As a result, general irritability of the nervous system, general neurasthenic disturbances, or localized neuroses, develop. In most all the instances we see general signs of disturbed nerve function wherever there are local neuroses. Men are also exposed to numerous other injuries that weaken the nervous system—for instance, alcohol, tobacco, excesses in venery, etc.

The nutrition of the patient is of less significance in the genesis of neuroses of the stomach. The disease occurs with equal frequency in strong and well-nourished men as in pale, anemic, and weakly men. Neuroses of the stomach, if they persist a long time, of course finally interfere with nutrition. In these instances, however, emaciation is not one of the predisposing factors of the gastric neuroses, but a consequence of persistent nervous dyspepsia.

Classification of Neuroses of the Stomach.—We have seen that neuroses of the stomach may present the appearance of some primary disease of the stomach, may be one of many symptoms of general nervous hysteria or neurasthenia, or may finally be reflex symptoms due to disease of some other organ. Clinically, these different forms cannot be sharply differentiated. If we undertook to discuss each form separately, we would have to repeat frequently. In this instance, as in so many other cases, the general symptom-complex, not the etiology, must be particularly emphasized. The clinical picture, aside from certain peculiar traits, will be the same in all these conditions; a variety of primary causes for the different forms, however, will lead us to treat each case differently.

Disturbances of the nervous functions may involve the secretory, motor, and sensory spheres. Sometimes they involve only one of these nerve areas, sometimes several, sometimes all of them. Quite frequently we notice that the symptoms alternate, so that on one day the symptoms point to a disturbance in one sphere, on another day in another sphere. These clinical peculiarities lead us to subdivide the neuroses of the stomach into neuroses of motility, secretion, and sensibility. We may expect *a priori* to find that deviations from normal may occur in either direction—that is, we may encounter abnormal irritability on the one hand, or depression of function on the other.

Before entering into a discussion of these different symptomatic expressions of perverted nerve function in the stomach I wish briefly to call attention to one other point. In describing the neuroses of the stomach it is impossible to do more than describe each one of these three forms—namely, neuroses of secretion, motility, and sensibility—separately.

This is the only way to give a fairly complete picture of the different variations that may occur. In reality, however, we rarely find these disturbances limited to one nervous sphere or to one function; more often disturbances of motility are associated with disturbances of sensibility, etc. Nervous hyperacidity or hypersecretion, for instance, is primarily a neurosis of secretion and irritative in character. We call hypersecretion a secretory neurosis because the dominating symptom is the increased secretion of gastric juice. This condition is almost always accompanied by symptoms of sensory irritation, hyperesthesia, gastralgia, nausea, and symptoms of motor irritation, as spasm of the pylorus, vomiting, etc. This may illustrate what I mean, for hypersecretion is a gastric neurosis in which symptoms of irritation are observed in the sensory, motor, and secretory spheres all at the same time. In other instances only a few disturbances occur together; in still other cases, though rarely, the nervous perversion is limited to one sphere and is manifested by a uniform train of symptoms. Even that form of nervous dyspepsia that Leube first described, and in which the disturbances in the sensory sphere were the most conspicuous, while digestion itself proceeded in a normal manner, is, strictly speaking, not a pure neurosis of sensibility, for in this condition we frequently encounter symptoms of motor irritation—as, for instance, vomiting. In the majority of cases, I may say as a rule, we see combined disease-pictures; in addition other symptoms may be added to those that are due to the nervous disorder of the stomach. The latter may either be due to the same primary cause as the nervous dyspepsia or they may be reflex symptoms of remote origin. Thus, headache, mental depression, lack of energy, a distaste for work, or, in other cases, a sensation of fear, palpitation, dyspnea, violent sweating, etc., are seen together with the symptoms of a neurosis of the stomach. The character of these morbid phenomena, the area that is involved in addition to the stomach, will depend on the constitution of the patient and the individual irritability of certain parts.

As soon as the disease-picture attains a certain severity it is frequently difficult to decide which of the symptoms are primary, which secondary. In almost all instances the syndrome "nervous dyspepsia" is merely the expression of some general nervous disorder, rarely of a primary local neurosis.

We see, therefore, that any description of neuroses of the stomach that limits itself to a discussion of the single forms of this disease must be deficient and incomplete in many directions. We encounter the same difficulty in attempting to describe, for instance, hysteria; for here, too, it is almost impossible to include all the numerous forms and combinations that we encounter in practice; all we can do is to describe the different disturbances that may be encountered in different areas. It is impossible to describe the innumerable variety of combinations that we may occasionally encounter.

One more remark in conclusion. Neuroses of the stomach, even if they persist for a long time, and even if the symptoms all point in one direction, rarely present the same picture during the whole period of the disease. Thus, nervous perversions of secretion frequently vary in intensity, so that the syndrome presented also varies. In addition, the neurosis may alternately involve the secretory, the motor, or the sensory sphere, so that in this way the syndrome again changes. As a matter of fact it may be considered characteristic of neuroses of the stomach that the syndrome presented varies so frequently and in so many ways. This aids us in differentiating these perversions from organic diseases of the stomach. We can usually assume that we are dealing with a neurosis if the symptoms appear in an altogether irregular manner, if they involve one sphere of the motor functions to-day, and another one to-morrow. Let me emphasize particularly that this change of symptoms is an important characteristic of nervous dyspepsia. Another peculiarity of neuroses of the stomach is that they frequently originate from general nervous disorders, and finally that they are amenable to treatment by psychic or suggestive therapeutics.

THE DIFFERENT FORMS OF GASTRIC NEUROSES.

(a) **Motor Neuroses of the Stomach.**—Neuroses of motility may occur in two forms—namely, an irritative and a depressive form. The first class includes hyperkinesis or hypermotility, peristaltic unrest of the stomach, spasm of the cardia, spasm of the pylorus, nervous belching, and nervous vomiting. Increased motor activity of the stomach is common to all these forms. It is, generally speaking, impossible strictly to differentiate all these manifestations, for the reason chiefly that one form may merge into the other. Spasm of the stomach, for instance, may, under certain circumstances, cause vomiting. The spasm may involve only the cardia or only the pylorus, or may involve, on the other hand, the whole organ. Sometimes the location of the spasm may vary in the same subject, so that in the beginning we see a spasm of the pylorus, and later gastrospasm and vomiting. At the same time the different forms that we have enumerated present certain distinctive points of difference.

Hypermotility of the Stomach.—Hypermotility or hyperkinesis of the stomach is merely an acceleration of the motor action of the organ. The motor function of the stomach is twofold—first, to cause an intimate mixture of the ingesta with the gastric juice; second, to propel the stomach-contents into the intestine. These two functions are performed by the peristaltic movement of the organ. As a rule, the stomach-contents is retained in the stomach until it becomes pultaceous in consistence, and is then propelled into the intestine. We speak of hyperkinesis—hypermotility of the stomach—whenever the stomach-contents is forced into the intestine with abnormal rapidity—that is, in an abnormally short time. We cannot say, however, that hypermotility is always necessarily a primary neurosis, for we frequently encounter hypermotility in certain organic disorders of the stomach, particularly in certain forms of achylia gastrica and in a number of diseases that are complicated by hyperchlorhydria. In the former class of cases we frequently find that the stomach propels the ingesta into the intestine much more rapidly than normal. This occurs as soon as the function of the glands becomes insufficient, so that the chemical conversion of the ingesta cannot occur in the stomach. It almost seems as though the loss of peptic power were compensated by the increase in the motor power of the stomach. In another group of cases—namely, in hyperchlorhydria—the chemical transformation of the ingesta occurs more rapidly than normal, owing to the increased secretion of gastric juice; consequently the ingesta are also propelled into the intestine more rapidly than normal. In neither case, therefore, are we dealing with a neurosis. We can, however, speak of a neurosis even in those cases of hyperchlorhydria or achylia gastrica that originate from some nervous disorder. Even here, however, we are not dealing with a motor neurosis, but with a secretory neurosis. In the former instance increased motility, or, better, abnormally rapid expulsion of the ingesta, is merely a sequel of nervous hyperchlorhydria; in the latter, so to say, a process intended to compensate the loss of peptic power. I do not doubt that the stomach-contents is occasionally propelled in an abnormally short time, and before the ingesta are sufficiently converted by direct nervous influences—that is, by direct irritation of the nervous apparatus governing the peristaltic movements of the stomach. This condition is analogous to the increased intestinal peristalsis that we frequently see, and that may occasionally be due to nervous irritation alone.

The only way to demonstrate the existence of hyperkinesis is to aspirate the stomach-contents. As a rule, this method will show us that the ingesta are propelled into the intestine sooner than normal. My own experience teaches me that in the majority of cases nervous hypermotility of this kind is primarily due to nervous hyperchlorhydria; in other words, that hypermotility is merely a sequel of hyperchlorhydria. The latter condition, unless complicated in some way, as a rule, leads to an early expulsion of the ingesta. If, therefore, the condition of hyperchlorhydria can be relieved, the hypermotility of the stomach usually disappears at the same time.

In the form of hyperkinesis that we have described the character of the motor action of the stomach is the same as under normal conditions; quantitatively, however, the function is perverted, for the muscular action is increased and the propulsion of ingesta occurs in an abnormally short time. Here too, therefore, the final purpose of the movements of the stomach—namely, the expulsion of the ingesta—is accomplished, only more rapidly at an earlier period than normal.

The diagnosis is based exclusively on the result of aspiration of the stomach-contents. In order to determine whether the hypermotility is primary or secondary; whether or not it is due to some secretory disturbance, the chemism of the stomach must be determined; in other words, the stomach-contents aspirated must be submitted to chemical analysis. Hypermotility *per se* does not produce any distressing symptoms.

Peristaltic Unrest of the Stomach.—The term "peristaltic unrest of the stomach" was originated by Kussmaul. The name implies that there is an unusually active peristaltic action of the stomach that, under certain conditions, may even become visible through the abdominal walls. Increased peristaltic movements of the stomach are most frequently found in stenosis of the pylorus or of the first portion of the duodenum. The cases that Kussmaul included under peristaltic unrest of the stomach were not due to stenosis; all the symptoms seemed to point to some morbidly increased irritability of the nervous apparatus governing peristalsis and leading to "peristaltic unrest"; in other words, his cases were pure neuroses of motility. The peristaltic movements appeared early in the morning, when the stomach was empty or contained only a little air. They were quite active even at this time. After eating they became very active, and sometimes even exceedingly violent. Unfortunately, Kussmaul failed to test the peptic powers of the stomach and the period of digestion in the two cases he reported. Other authors who reported cases after him also failed to give us any detailed information in regard to the duration of digestion,—that is, whether it was normal, prolonged, or abbreviated—as we did in the cases of hypermotility that were previously discussed.

Both of Kussmaul's cases were at the same time afflicted with gastroptosis, so that it was possible actually to see the peristaltic movements that the stomach was performing. In both cases the appearance of peristaltic unrest was preceded by serious injury to the nervous system; the one case experienced a very depressing emotional shock, the other case had abused his constitution by sexual excesses.

In searching for the cause of this peristaltic unrest in any given case we must primarily exclude the existence of a mechanical obstacle at the pylorus or its vicinity. These mechanical forms can readily be differentiated from the nervous ones, for in the former increased peristalsis is seen only when the stomach is full, in the latter as well when the stomach is empty.

There are, however, cases of nervous peristaltic unrest in which the abnormal movements of the stomach are not continuous, as in Kussmaul's cases, but appear at intervals. Peristaltic unrest is different from hyperkinesis, which we have previously mentioned, in this respect, that peristalsis is very active in the former condition and is experienced as a disagreeable sensation by the patients themselves, and may even occur when the stomach is empty. It is almost impossible, however, to differentiate these two forms distinctly. It is more conservative to remember that the two are different merely in degree, and that numerous transition-forms between the two exist. In a case of hyperchlorhydria with hypermotility, for instance, it may be difficult to decide whether the painful and disagreeable sensations are due to hyperesthesia or to increased peristalsis. In a great number of cases of peristaltic unrest of the stomach disturbances of motility are probably not the primary factor. As a rule, we find that these motor disturbances are secondary and due to hyperchlorhydria. This, however, applies only to those cases in which increased peristalsis appears only during the time of digestion. In Kussmaul's cases hyperchlorhydria certainly was not the cause of the peristaltic unrest. We know this even though the gastric juice was not analyzed, for in his cases peristalsis appeared when the stomach was empty. It is hardly probable that hypersecretion was present, although we might think of this condition in view of the fact that the subjective symptoms were so intermittent. In cases that present the characteristics of Kussmaul's cases we are forced to assume that the motor nerves of the stomach are abnormally irritated.

That the spasm in these cases did not only involve the circular musculature of the pylorus was shown by the very active peristaltic movements that visibly occurred all over the stomach.

If the stomach is normal in size and situated in its normal position, and if the patients are well nourished, peristaltic movements of the stomach, however active they may be, can hardly ever be seen. If, on the other hand, the stomach, as in Kussmaul's cases, is dislocated or ectatic; if the abdominal walls at the same time are very thin—these abnormal movements can be seen directly without difficulty. Throughout the stomach region alternate protrusions and retractions are seen. These movements may become so active that the stomach appears contracted like an hour-glass stomach; or, in cases, wave-like movements can be seen that usually extend from the left above to the right below, from the fundus to the pylorus. They rarely run in an opposite direction; in other words, the peristalsis is rarely reversed. Occasionally a loud succussion-sound can be heard at some distance, particularly if the stomach contains fluid and air together. The peristaltic unrest may extend even to the duodenum.

If the stomach is in its normal position and is not enlarged, and if the abdominal walls contain much fat, we must rely on the statements of the patient alone for the diagnosis. They complain of an almost continuous feeling of unrest and burrowing in the region of the stomach; they claim to feel active, wave-like movements and retractions in the stomach region, and complain that these abnormal sensations persist uninterruptedly. Quite frequently there is loss of appetite after a short time. Attacks of nausea and vomiting may also occur. The permanent unrest in the stomach usually causes the patient to lose much asleep.

The diagnosis is easy in those cases in which the stomach is dislocated or dilated, so that the active peristaltic movements can be seen directly; less easy in cases in which the stomach is in its normal position and normal in size, as the diagnosis must be based on the subjective complaints of the patient alone.

If the existence of peristaltic unrest can be demonstrated, it must be determined whether or not it is due to some mechanical obstacle at the pylorus or in its vicinity. As a rule, however, ectasy will be found in the latter class of cases, and peristaltic unrest will usually appear only when the stomach is full. The presence or absence of hyperchlorhydria or of an abnormal development of gas in the stomach can be determined only by analyzing the stomach-contents. This method of diagnosis, therefore, should never be omitted.

The diagnosis, "nervous peristaltic unrest," should be made only if the last-named causal factors can positively be excluded, and if, at the same time, a certain nervous predisposition can be discovered, or if the history of the case reveals that the nervous system was damaged in some way before peristaltic unrest was complained of.

Peristaltic unrest of the stomach should rarely be confounded with a similar condition of the intestine, for involvement of the intestine can usually be excluded by determining the boundaries of the stomach —if necessary, with the aid of inflation. Peristaltic unrest as a primary neurosis of the stomach is comparatively rare; as a secondary manifestation of a variety of diseases of other organs, however, it is quite frequent.

The course of the disease will depend on the primary cause. If the latter can be discovered and removed, the course will be shorter than if this cannot be done.

A prognosis cannot, as a rule, be made, as it is altogether dependent on the primary cause of the disease, the constitution of the nervous system of the patient, and many other factors.

Treatment should be directed primarily against the cause. If a gastric neurosis is the result of mental overexertion, of overirritation of the nervous system, of grief, sexual excesses, etc., we can expect good results from treatment only if we can succeed in removing these primary factors. The nervous system should be strengthened by hydriatic and other measures; the diet should be carefully regulated to suit the case; all mechanical, chemical, and thermic irritants to the stomach should be avoided; the diet should be strengthening but digestible, non-irritating, and not too voluminous. Very little should be eaten at supper-time.

Various direct local remedies have been recommended. In some cases cold applications to the stomach, in others warm ones, have been found useful. The application of galvanic electricity is worthy of trial; the current may be applied percutaneously or intra-abdominally, the anode being placed in the region of the stomach or introduced into the stomach. If the electrode is introduced into the stomach, it is usually well to precede it with a small quantity of physiologic salt solution. If the patients are very much annoyed by peristaltic unrest, certain sedatives may be administered internally—for instance, sodium bromid, belladonna, codein, etc.

Hyperkinesis and peristaltic unrest of the stomach are forms of spasm in which the motor function of the stomach as a whole is increased. There are, however, other forms of spasm that involve only single portions of the stomach or certain portions of the gastric musculature. To the latter class belongs spasm of the cardia and of the pylorus.

Spasm of the Cardia.—Spasm of the cardia consists in a convulsive contraction of the circular musculature of the cardia, which is usually accompanied with violent pain and may last for a varying period of time. Such spasms of the cardia are occasionally observed when the stomach-tube is introduced, and we occasionally see this instrument held fast in the cardiac region. Again, we may observe the spasm after rapid eating, or after the introduction of very hot or very cold drinks, or after swallowing coarse, hard morsels of food, etc.

We also see spasm of the cardia in certain organic diseases—for instance, in ulcer or carcinoma of the cardia. Even in simple hyperchlorhydria spasm of the cardia occasionally develops, though less frequently than spasm of the pylorus. In certain affections of the stomach that are complicated by an abundant development of gas there may also be spasm of the cardia. In the latter instance, however, the spasmodic contraction is not limited to the cardia, but is seen also in the pylorus.

Subjects who have acquired the pernicious habit of swallowing large quantities of air may also develop spasm of the cardia and the pylorus, and in this way produce distention of the stomach. This distention may be so great that the diaphragm is forced upward, and difficulty in breathing results in this way.

Spasms of the esophagus and cardia are quite frequent in tetanus, as in hysteria, neurasthenia, and after violent psychic shocks.

Spasm of the cardia, therefore, may either be a sequel of a variety of diseases or a primary condition. In a proportion of cases the spasmodic attacks are short and recur at long intervals ; in other cases the condition is very obstinate and chronic, and recurs with great frequency.

As a rule, an attack of cardiac spasm occurs suddenly and unexpectedly. The patient may be in perfect health, when suddenly a painful, spasmodic sensation is felt in the region of the cardia that radiates upward toward the chest, and in some instances toward the back. If the spasm occurs at the precise moment when the patients are swallowing a morsel of food or some liquid, they feel distinctly that the ingestum is arrested.

A spasm of this kind may stop after a few minutes or may persist for a longer time, or finally may recur many times. Occasionally there is violent gagging, with or without vomiting. The spasm usually persists for a considerable time if it occurs during eating, and no amount of gagging or vomiting seems to relieve the spasm.

In mild degrees vomiting occurs within a short time, so that the food or fluid, usually mixed with much mucus, is evacuated. In severe cases the attacks of spasm recur with such frequency that it is difficult to administer any food whatever to these sufferers.

We have mentioned above that swallowing air, or the existence of fermentative processes in the stomach that lead to the abundant development of gas, may produce spasm of the cardia. This distention of the stomach, so-called pneumatosis, usually leads to spasm of the pylorus at the same time.

Primary spasm of the cardia, however, is unable to produce distention of the stomach. That this is the case can be easily determined by inflating the stomach artificially. If the pylorus is insufficient the stomach is never distended, and the distention of the organ persists only as long as the cardia and the pylorus remain closed.

Some authors claim that a secondary result of these frequent spasms of the cardia is dilatation of the lower portion of the esophagus above the area of spasmodic contractures. They claim that consequent to this stagnation occurs, and finally secondary changes in the mucous lining of the esophagus. Personally, I consider this problematic. The inverse sequel of events, however, is frequently observed, for inflammatory changes and diverticulation of the esophagus may, in the first place, lead to stagnation, and in the second place to spasm of the cardia.

The diagnosis is easy in those cases in which spasm of the cardia is transitory and occurs at irregular intervals, and in which the act of deglutition can be performed without difficulty in the interim. If acute tympanites occurs, there must be spasm of the pylorus at the same time. The cause of the condition is frequently difficult to determine. If certain nervous disorders are present, and if the spasm occurs after psychic disturbances, the origin of the condition is at once revealed.

Chronic forms of spasm of the cardia that have a tendency to recurrence are more difficult to diagnose, for carcinoma of the cardia or ulcer of the cardia may produce similar symptoms. As a rule, however, a correct diagnosis can be made, provided we study the case with sufficient care. In the last-named processes the obstacle seems to be more uniform in character. It is more easy to pass thin sounds than thick ones, whereas in spasm of the cardia the reverse is the case. If traces of blood are raised with the sound, this speaks for ulcerative processes. If there is increasing cachexia, swelling of glands, a gradual development and exacerbation of the symptoms of stenosis, and if the patient is advanced in years, all these features speak for carcinoma and against spasm of the cardia. The differential diagnosis between diverticula that are situated low down or congenital dilatation of the esophagus and spasm of the cardia should be easy.

Treatment is primarily prophylactic. If the mucous membrane of the esophagus is very irritable, and if this irritability is the primary cause of the spasm, if the spasm occurs particularly after eating and drinking, the patients should be advised to eat slowly and to masticate their food thoroughly, and to avoid drinking liquids that are very hot or very cold. A very good method of treating this increased irritability of the esophageal mucosa is to institute a course of sounding with thick sounds that should be left in place for some time. This probably is the best means to reduce gradually the condition of hyperirritability. If the spasm of the cardia is due to erosions of the cardiac region, the passage of sounds is contraindicated. In these cases the parts should be placed at rest as much as possible, and fluid or pultaceous, non-irritating food administered. In severe cases alimentation by mouth should be replaced by rectal feeding continued for a long time. If the spasm of the cardia is due to an abnormal development of gas, this condition must be combated, and if necessary stagnating stomach-contents removed by lavage. Patients who are in the habit of swallowing much air should be taught to get rid of this habit.

The best treatment for nervous spasm of the cardia *per se* is, as we have said, the methodic introduction of thick sounds. Occasionally internal galvanization of the stomach performed as above may be of some use; it is certainly worthy of trial.

Spasm of the Pylorus.—Spasm of the pylorus is a very frequent occurrence, but is nearly always a secondary condition. Spasm involving the pylorus is seen in hyperchlorhydria, in acute and chronic gastrorrhea, in round ulcer, in irritation of the stomach by indigestible diet or by food that is too hot or too cold, or by strong spices. Occasionally coarse morsels of food and certain articles of diet may irritate the stomach mechanically, and in this way lead to spasm. Spasm of the pylorus is seen particularly in those conditions of the stomach that are complicated by an increased production of hydrochloric acid, less frequently in carcinoma, in which the production of gastric juice is reduced, even though in this disease an abundant quantity of organic acid is formed.

There is a great diversity of opinion in regard to the existence of primary spasm; it is questionable, in fact, whether or not primary spasm of the pylorus ever occurs as a genuine motor neurosis. It is certainly conceivable that such a condition may exist; but if it does, it is certainly rare. Before we are justified in considering a spasm of the pylorus as a genuine primary motor neurosis, we must be able to exclude all perversions of gastric secretion, all mechanical obstacles in the pyloric region, all adhesions, cicatrices, etc. The clinical symptoms produced by spasm of the pylorus are, in the first place, pain; in the second place, increased peristalsis of the stomach; in the third place, under certain circumstances, vomiting.

If the spasm occurs at a time when ingesta are still present in the stomach, the propulsion of the ingesta is delayed. The longer the spasm lasts the longer must the ingesta remain in the stomach, provided they are not evacuated by vomiting. Secondary spasm of the pylorus of this character, particularly if it recur with great frequency, undoubtedly may lead to motor insufficiency, and finally to ectasy of the stomach. I refer to the section on the Continuous Secretion of Gastric Juice for a more detailed description of this sequence of events. I doubt whether primary nervous spasm of the pylorus alone can ever cause the development of these conditions. I do not remember a single case of ectasy in which it seemed even probable that the enlargement of the stomach originated in this way.

Nervous Belching (*Eructatio Nervosa*).—In certain pathologic conditions of the stomach that are characterized by abnormal fermentation and the development of abundant quantities of gas, the patients complain of frequent belching. Even healthy subjects are forced to raise a good deal of gas from the stomach as soon as they drink large quantities of carbonated beverages, champagne, etc. The same applies to subjects who eat very rapidly and who do not masticate their food thoroughly, and who consequently swallow a great deal of air. This air, too, must be removed by belching. A little air is always present in the stomach, for even under normal conditions some air is always swallowed. A healthy subject, therefore, may have to belch occasionally, particularly if he introduces large quantities of gas with the beverages he drinks. If the stomach is diseased, and if abundant fermentation and development of gas occur within the organ, belching may become excessive.

In this discussion we are not dealing with cases of this kind, but with cases in which belching occurs frequently and at short intervals, and is altogether independent of the character of the food. Belching in these cases is of purely nervous origin. As a rule, it is noisy. It may persist for hours or even days, or may occur in paroxysms. Frequently it is caused by some psychic shock. The condition is particularly frequent in hysteric women and in neurasthenics. Occasionally it is observed in children. The gas raised consists essentially of atmospheric air that may have entered the stomach either by aspiration or by swallowing.

Some investigators have objected that air cannot be swallowed, chiefly because, as they claim, the pressure in the stomach is greater than in the esophagus and in the thoracic cavity, so that air can enter the stomach only if the patient should intentionally swallow it. As a matter of fact, they claim that patients never do this. I think, however, that air may undoubtedly be forced into the stomach even without visible efforts at swallowing. Oser has called attention to a second possibility—namely, that air may occasionally be aspirated into the stomach without any effort at swallowing. He claims that the stomach constitutes an elastic sac that can be compressed, but always has a tendency to return to a position of tensile equilibrium. He assumes that contraction of the circular fibers of the stomach empties the organ, and that contraction of the longitudinal fibers opens it again and establishes a lumen within the stomach. In the latter act the cardia is opened so that air enters the stomach from the esophagus even without any effort at swallowing. As soon as the organ contracts the air is expelled.

Bouveret considers spasm of the pharynx essential for swallowing air. This pharyngeal spasm, he claims, produces spasmodic deglutition.

Those cases of belching that follow an abnormal development of gas in the stomach, or that occur when abundant quantities of gas are swallowed in different beverages, naturally do not belong to the group of nervous belching. In these cases the gas belched is probably air that has been swallowed. Undoubtedly these patients could avoid much of the belching if they would only try, as much of the air is expelled by a *voluntary* effort on the part of the patients. If the attention of the patient is abstracted, or if some psychic shock is administered by the physician, it is almost always possible to stop the belching, usually, however, for a short time only.

A variety of factors may be considered the direct cause of this nervous form of belching—namely, psychic shock, fright, fear, etc. A certain nervous predisposition, an hysteric or a neurasthenic tendency, must, however, exist at the same time. Occasionally this nervous disorder has been seen to occur in a number of persons who lived together. Children in particular show a tendency to belch if they see another child belching.

Spasmodic clonic contractions of the stomach undoubtedly have something to do with these eructations. The cardia, of course, must be open, or at least the normal closure of the cardia released for a time. In many cases possibly air does not enter the stomach but merely enters the esophagus (Bouveret). The diagnosis of eructation is easy if the patient can be seen in an attack. In order to determine whether we are dealing with the nervous form or with abnormal development of gas in the stomach, the stomach-contents should be analyzed and a fermentation-test performed. If abnormal fermentation can be discovered, the possibility of a nervous origin of the trouble is excluded. The course of the disease, the persistence of the eructations, their cessation after psychic shock, are all valuable clues in diagnosing this nervous form.

Treatment should be practically psychic. I have repeatedly seen cases in which the disease persisted for months notwithstanding the administration of all imaginable forms of medication, but in which suggestive treatment carried out in the hospital stopped the attacks in a short time. I do not wish to deny that certain drugs may occasionally be of value, but I am very much inclined to the opinion that they act more or less by suggestion. The remedies that have been recommended are preparations of the bromids, arsenic, and belladonna. In some instances climatic cures, hydrotherapy, and similar methods may aid in raising the tone of the nervous system.

Nervous Vomiting (*Vomitus Nervosus*).—So-called nervous vomiting is not a true gastric neurosis. This is evidenced by the fact that other organs, for instance, the abdominal muscles and the diaphragm, are involved at the same time; the latter, in fact, are chiefly concerned in the act of vomiting. It would lead us too far to enter into a detailed analysis of the mechanism of vomiting in this place. I merely recall the fact that for a time the belief was prevalent that the stomach itself had nothing whatever to do with vomiting. The reason for this was that Magendie succeeded in replacing the stomach by a pig's bladder, and nevertheless could produce vomiting. The conclusion was drawn from this experiment that the stomach had nothing to do with vomiting; but this view was soon shown to be wrong. It may be considered established, however, that the act of vomiting is not due to the action of the stomach exclusively. Other factors that are principally concerned in this act are the contraction of the abdominal muscles and the fixation of the diaphragm; in addition, the contraction of the stomach accompanied by closure of the pylorus and opening of the cardia. We see, therefore, that vomiting is brought about by the synergetic action of different muscles, both voluntary and involuntary. The stomach at the same time is actively concerned in the process, and for this reason I feel justified in including nervous vomiting under the neuroses of the stomach.

Those cases in which vomiting is due to some organic disease of the stomach do not, of course, belong to this category. Vomiting, as we all know, is a frequent symptom of a great variety of diseases of the stomach. In this place we will limit ourselves to discussing the purely nervous form of vomiting, that may either be cerebral or spinal in origin, or may be due to some reflex disturbance.

Cerebral vomiting is observed in a number of organic diseases of the brain and its meninges, in anemia and hyperemia of the brain, in concussion of the brain, in tumors of the brain, and in similar conditions, also in certain intoxications, as by opium, morphin, chloroform, ether, and tobacco. The vomiting of uremia and cholemia may also be considered due to intoxication. In all these forms vomiting is merely an isolated symptom that is observed only occasionally, so that it is hardly worth while to enter into a detailed discussion of this form of vomiting. Vomiting following psychic shock is more interesting and merits particular discussion. Disgust, anger, and fright, as we know, may frequently cause loss of appetite and even vomiting; the same applies to psychic excitement, violent emotional shock, and mental overexertion.

Von Leyden has described a peculiar form of periodic vomiting that resembles somewhat the gastric crises seen in tabes. Periodic vomiting, however, is a more independent disease, and seems to stand in no relation whatever to lesions of the spinal cord. The onset of the disease is usually sudden. As a rule, no cause can be discovered, or it may occur after a simple attack of indigestion, some psychic excitement, or a cold. The attacks recur at irregular intervals. Each attack lasts for several hours or may persist for from one to several days. There is usually a feeling of excessive nausea, followed by violent and copious vomiting; at the same time the patients complain of general discomfort, and occasionally of violent headache. As soon as the patients eat anything violent vomiting ensues. The material raised may be so abundant that it seems to be more bulky than the amount ingested; finally bile is vomited. As a rule, this occurrence terminates the attack. Occasionally, however, it may continue for several days or longer; finally the stomach quiets down, the attacks of vomiting recur less frequently, and a little food can be borne; at the same time sleep becomes possible and general improvement sets in. In a short time the patients are completely restored to health and can eat anything, until ultimately another attack occurs.

Von Leyden recommends rest, ice, morphin, belladonna, and hyoscyamus.

This neurosis of the stomach is unquestionably analogous in many respects to the gastric crises seen in tabetic patients.

In searching for the cause of this disease we find that only a few of the cases reported could be attributed to organic disease of the nervous system. In many of the cases no connection whatever could be discovered with diseases of the spinal cord; the attacks occurred like violent attacks of migraine, and seemed to be influenced by emotional disturbances both in regard to the frequency with which they occurred and their violence. In some instances it appeared as if the whole trouble were due to indigestion, and in still other cases the primary cause seemed to be located in the uterus.

[The periodic or cyclic vomiting in children, which during the past ten years has much attracted the interest of American observers, seems to be somewhat different from the affection which von Leyden describes as occurring in adults. The cyclic vomiting in children usually appears when the infant is two or three years old, and gradually passes off, although it sometimes continues until after puberty. Among the best articles on the subject are those by Snow,[1] Whitney,[2] Blodgett,[3] and Crozer Griffith.[4] There have also been contributions by Gee, Rachford, Holt, Boas, Einhorn, and others. This group of cases follows a rather definite course. From a condition of apparent health the child becomes suddenly languid, has a moderate elevation of temperature, accompanied by vomiting that is persistent and sometimes violent. There soon develops prostration, often of an alarming kind, and a fatal issue of the case seems not improbable. As suddenly as it began the vomiting ceases, and the child is rapidly restored to health. The interval between the attacks varies,—sometimes a few weeks, sometimes several months. Occasionally it seems to have a definite cycle or period. After the stomach is free from food the vomited substance appears to be pure gastric juice, and in several cases that have come under my observation there has been a high degree of acidity. This high acidity decreased toward the end of the attack, and sometimes has been followed by a temporary depression of the acidity of the gastric secretion. The remarkable features in this disease are the early appearance of a grave prostration and the promptness with which this prostration disappears when convalescence is established. In a recent monograph on the subject by Soleis, of Paris, the views expressed are that the affection depends upon some disturbance in metabolism, associated with the excessive development of uric acid; but it is probable that the disturbance in metabolism is a mere accompaniment, and not a cause of the vomiting. A number of recorded cases have terminated fatally.

No plan of treatment has been rewarded by great success. The most practical suggestion is that of introducing water by hypodermoclysis or enteroclysis, as recommended by W. W. Johnson and Kinnicut. Slight improvement from lavage is occasionally seen, and sedatives have apparently been of temporary use. In one of my cases benefit seemed to follow the administration of a full dose of calomel in the early hours of the attacks, before the vomiting was fully established.—Ed.]

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[1] *Trans. of Am. Ped. Soc.*, Dec., 1893.

[2] *Arch. of Ped.*, 1898.

[3] *Boston Med. and Surg. Jour.*

[4] *Trans. of Assoc. Am. Phys.*, 1900.

Spinal forms of vomiting are rare. An example of this class are the gastric crises that occur in tabes. In an attack of this character the patients experience sudden pain that usually rises on both sides of the abdomen as far as the epigastric region, and seems to persist in this area; at the same time very obstinate and very distressing vomiting occurs, during which food is at first vomited, and later a fluid containing much mucus. These attacks are also occasionally accompanied by secretion of gastric juice. The gastric crises of tabes usually persist without interruption for two or three days, and in the intervals between the attacks the functions of the stomach are altogether normal. The crises may appear in the initial stages of the disease, and do not necessarily stop when the ataxic stage develops. In some instances they persist throughout the whole course of the disease until the death of the patient. Gastralgic attacks of this kind have occasionally been seen in other diseases of the spinal cord. Charcot states that he saw them in cases of general paresis and in the course of multiple sclerosis; von Leyden observed gastric crises in a case of subacute myelitis.

The attacks of vomiting that occasionally occur in cases of neurasthenia and hysteria must also be included in the group of central vomiting. These attacks are distinguished from the attacks of periodic vomiting described by von Leyden by their great irregularity and their varying character. In many instances the attacks of vomiting are not preceded by nausea, but occur immediately after the food is swallowed. Some patients vomit solid material alone, others only liquids; some patients vomit anything they may eat if they swallow it while sitting up, but can assimilate and retain the food if they swallow it when lying down; still other patients finally vomit only certain articles of food. The digestibility or indigestibility of the different articles of diet seems, however, to have nothing to do with these phenomena. As a rule, as I have said, vomiting occurs very easily, and is not preceded by nausea. It is peculiar that the general nutrition of these patients rarely suffers even though vomiting persists for a long time; in some cases, however, emaciation and loss of strength supervene after a time.

Nervous vomiting is less frequent in neurasthenics than in hysterics. Rosenthal claims to have seen nervous vomiting quite frequently in neurasthenics, and states that in these subjects the lower thoracic and upper lumbar vertebræ were very sensitive to electric irritation. In all of these cases the mucous lining of the stomach is very hyperesthetic, so that the suspicion seems justified that the vomiting is secondary—that is, the result of increased sensibility and irritability of the gastric mucosa. It has been stated that this form of vomiting is most common in sexual neurasthenics.

Occasionally children develop nervous vomiting if they overexert themselves at school. It is possible that frequent masturbation can be made responsible for the increased nervous irritability in many of these children.

As a rule, nervous vomiting is reflex in character. It may be due to disease of almost any organ. In some subjects the mere sight of some disgusting thing, a disgusting smell, looking at high waves, etc., may cause vomiting. The attacks of vomiting that are seen in neuralgia of the trigeminus must also be considered reflex. Reflex vomiting is particularly apt to follow disease of the organs of the mouth and pharynx by direct transmission of reflex irritation through the vagus. Disease of the abdominal organs, particularly if the peritoneum is involved, also plays an important rôle in this respect. Acute and chronic peritonitis, typhlitis, and incarcerated hernia, almost always lead to reflex vomiting. As a matter of fact, vomiting is usually one of the first symptoms of these conditions. Another important and frequent source of reflex vomiting is disease of the female sexual organs. Even under physiologic conditions vomiting occasionally occurs at the time of menstruation, and particularly at the time of pregnancy, more frequently, of course, if some pathologic condition exists in the sexual sphere. It must be remembered, however, that vomiting occurs in only a limited number of women, and that there are a great many women who are never afflicted in this way even during menstruation, during pregnancy, or if the sexual organs are diseased.

In order that reflex vomiting should occur the patients must be particularly predisposed, must suffer from a particular irritability of certain nerve-tracts in addition to some change in the sexual organs. Many of the forms of pernicious vomiting of pregnancy, the *vomitus gravidarum*, undoubtedly belong to this group, for these attacks are also due to reflex irritation from the sexual organs. I have in the Introduction called attention to the fact that in the majority of cases a central predisposition exists in all these patients, and that this can be chiefly made responsible for the attacks of vomiting. I mentioned several cases in which vomiting that resisted all other means could be stopped by psychic or suggestive treatment. Nothing whatever was done to correct local disorders; the treatment was exclusively psychic.

Reflex vomiting following sexual disorders is less frequently seen in men.

Attacks of reflex vomiting can also be due to disease of other organs, notably of the liver, the kidneys (particularly in gall-stone or kidney colic, floating kidney), and of the intestine, particularly if parasites or foreign bodies are present, or if there is meteorism or obstinate coprostasis.

Diseases of the respiratory organs rarely lead to reflex vomiting. In a patient who possesses a particular predisposition to these attacks, disease of any organ may, of course, produce vomiting. Troeltsch, for instance, has called attention to the fact that irritation of the external auditory canal may occasionally lead to vomiting. In order that vomiting should occur, however, I repeat, a certain predisposition must exist —that is, certain nerve-tracts must be abnormally irritable.

I have mentioned that reflex vomiting is much more frequent in women than in men. In a general sense, any attack of vomiting is nervous in origin and all vomiting is reflex. We can only speak of a neurosis of the stomach, however, in those cases in which such inflammatory changes, as, for instance, a violent pharyngitis that causes vomiting, can be excluded, and in which the attack of vomiting is exclusively due to abnormal irritability of certain nerve-tracts.

In rendering the diagnosis of nervous vomiting, we must first exclude all organic changes of the stomach, and, second, determine the primary cause of the attack. Even if a neurosis of the stomach is present in any given case, we may still find that vomiting is secondary, and that the primary cause of the emesis is nervous hyperchlorhydria or hypersecretion. The only way to determine whether or not this is the case is to analyze the stomach-contents.

In order to determine whether or not the attack of vomiting is a complicating symptom of locomotor ataxia, we must study the peculiar characteristics of these tabetic crises, examine the tendon reflexes, the pupillary reactions, the general sensibility, etc.

In order to determine whether intermittent hypersecretion can be made responsible for the attack, the vomit must be carefully analyzed and the general course and character of the attack studied.

In many instances the symptoms of the attack are so typical that they at once suggest a neurosis. In hysteric cases, for instance, we frequently see the patients vomit immediately after swallowing even a small morsel of food; sometimes vomiting follows the act of swallowing so rapidly that we are almost forced to assume that the morsel did not even enter the stomach, and that vomiting was directly due to some perversion of the innervation of the esophagus, the so-called esophagism of French authors. I do not doubt that many cases of habitual nervous vomiting do not originate in the stomach, but are caused by spasm of the esophagus.

Another factor that speaks in favor of the nervous character of vomiting is the facility with which food is raised almost immediately after it is swallowed. The quality and the quantity of the food seem to be altogether indifferent and without influence. I have mentioned that some patients vomit all the liquids they swallow, whereas others vomit all the solid food. Whenever we see this we must think of a neurosis, particularly if certain psychic influences, the mood of the patient, etc., seem to exercise a distinct effect on the frequency of the attacks.

Still another factor that indicates nervous vomiting is the occurrence of vomiting only at certain times, usually immediately after some psychic shock or excitement. This view will be strengthened if we learn that the patient's appetite is normal and his digestion excellent in the intervals between the attacks. If, in addition, the general health is not impaired even though frequent attacks of vomiting occur, and if the patient develops no general dyspeptic disturbances, we can feel reasonably certain of the diagnosis.

The diagnosis of periodic vomiting where it occurs at long intervals is considerably more difficult. Von Leyden, who described these attacks, called attention to their similarity with gastric crises. The differential diagnosis between the two is easy, however, if we can determine the absence of initial symptoms of locomotor ataxia on the one hand, and can establish that the attacks occur with great regularity on the other. The condition cannot be confounded with hypersecretion or nervous gastroxynsis if the vomit is carefully analyzed.

If the diagnosis is established, we must next attempt to discover the cause of the trouble. In some instances it is self-evident; in others it is exceedingly difficult of detection, and in a number of cases we fail to find a primary cause. In pernicious vomiting of pregnancy the causal diagnosis is, of course, simple. The same applies to those attacks of vomiting that follow psychic shock or some violent emotional disturbance. Even though we can show, however, that the sexual organs are diseased or that the attacks of vomiting are in some way related to pregnancy, we do not, of course, understand the connection between the two, for the pregnant uterus or the anteflexion of the uterus does not *per se* cause vomiting. There must be another factor—namely, some abnormal irritability. The cases that Kaltenbach reported, and that we have mentioned above, demonstrate my meaning very clearly, for in his cases the attacks of vomiting stopped even though pregnancy continued. In his cases, moreover, the attacks were exceedingly obstinate, and still could be arrested by simple suggestion, as, for instance, that an indifferent procedure would stop the vomiting; or by some violent psychic shock, as in the case reported where vomiting stopped as soon as the woman began to worry about her husband, who had suddenly developed a serious disease; or, finally, by the fear of some procedure that was to be undertaken.

In treating genuine nervous vomiting we encounter many difficulties. Numberless methods have been recommended, but, as a rule, all of them fail. This is not surprising, for the only cases in which the treatment is really rational are those in which some local disease can be regarded as the primary cause of the vomiting, and in which this primary disorder can be cured. In many of the cases no direct cause can be discovered, so that causal treatment is essentially impossible. It would be altogether superfluous to discuss all the means and methods that we employ in the treatment of diseases of the brain and spinal cord, in diseases of the peritoneum, and in gall-stone and kidney colic, nor can we enumerate the methods that we employ symptomatically for the attacks of vomiting that occur in all these conditions. All we can do to stop vomiting in these cases is to combat the primary disease to the best of our ability.

If the patient is a person with a nervous disposition, and if mental overexertion, psychic excitement, emotional disturbances, and similar factors produce nervous vomiting, all these unfavorable influences must be eliminated as much as possible. If vomiting is one of the symptoms of hysteria or neurasthenia (and these cases merit our particular attention in this place), these primary neuropathic tendencies must be combated. We must treat the loss of nerve tone, the neurasthenic and hysteric tendency, by appropriate methods; for instance, hydrotherapeutic procedures, etc., preferably in a sanitarium. The most important thing is to stop the attacks of vomiting, for they constitute the most prominent symptoms of the disease. I believe that purely nervous or purely hysteric vomiting can be best relieved by suggestion. Some authors claim good results from one remedy, others from another, but the effect of suggestion is never altogether excluded.

I have mentioned that pernicious vomiting of pregnancy may also occasionally be cured by this means.

If we discover some disease of the sexual organs, or a floating kidney, we must remember that vomiting may be due to reflex irritation starting from these organs. In cases of this character, therefore, local treatment is indicated. Quite frequently we see the symptoms disappear after appropriate treatment of this kind. As a rule, however, the good effects are transitory, for anteflexion of the uterus or a floating kidney *per se* does not produce nervous vomiting; they can only do this in patients who are suffering from a particular predisposition to this disease. A cure of the local disease removes the primary cause of the vomiting, but not the predisposition.

If we fail to find a direct primary cause, or if treatment directed against such a cause is without effect, and if vomiting persists with great obstinacy, we should attempt to reduce the irritability of the stomach. The patients should be instructed to remain on the back, and to eat nothing but bland food. In very obstinate cases the stomach should be placed altogether at rest for a number of days and the patient fed exclusively by rectum.

Lavage of the stomach has been recommended by a number of clinicians, but I have failed to see good results from this procedure in the primary genuine form of nervous vomiting. I have seen good results, however, in cases in which the vomiting was secondary and directly due to hyperchlorhydria or intermittent hypersecretion.

Some observers claim to have obtained good results from irrigation of the stomach with nitrate of silver solutions.

Before prescribing a diet we must experiment a little. Some patients can take only certain articles of food; some prefer solid, others fluid nourishment. It is altogether impossible to formulate any definite dietary regulations; all that we can say in general is that the patients should limit themselves to small quantities of food at each meal.

The most popular remedies are morphin, opium, belladonna, and codeia, that had best be given in the form of suppositories. Morphin may also be given subcutaneously.

Internal remedies that have been recommended are preparations of the bromids, chloral hydrate, menthol (2 to 120 aq., 30 alcohol or brandy; dose, a tablespoonful), chloroform (3 to 6 drops on sugar or in water), and silver nitrate (0.3 to 100; a tablespoonful several times a day on an empty stomach).

A peculiar form of vomiting is the vomiting of blood that is occasionally seen in hysteric subjects. The same is sometimes seen in neurasthenic men, and is usually attributed to some central nervous disorder. Hemorrhages of this kind may recur frequently, and occasionally continue for some time; in other cases they recur at long intervals. The general health is rarely impaired by these hemorrhages. It is usually difficult to decide whether the hemorrhage is hysteric or whether it is due to some organic lesion. The absence of gastric symptoms proves nothing; the presence of hysteric symptoms is equally valueless. Personally, I have had no experience with this form. As the symptom has been mentioned by reliable observers (Stiller and others), I consider it my duty to mention it here.

The second group of cases that we must discuss are motor neuroses of the stomach that are characterized by a depression of the motor function. To this group belong atony of the stomach (gastroplegia), insufficiency of the pylorus and the cardia, rumination, and regurgitation.

Atony of the Stomach.—We have already discussed the form of motor insufficiency that is due to a reduction in the tone of the gastric musculature, to atony. We have given all that is known in regard to the etiology, symptomatology, and therapy of this condition in the section on Motor Insufficiency and Ectasy of the Stomach. This atony may complicate a variety of gastric disorders, but may also be the result of purely nervous disturbances. Atony of the gastric musculature may occur after certain injuries, just as paresis of the intestinal muscularis may occasionally be seen after some sudden psychic shock.

Atony of the stomach may also be one of the symptoms of certain diseases of the central nervous system. When it originates in this way its duration varies; it may persist for a long time or may be very transitory.

It remains undecided whether that form of atony that is caused by overloading the stomach with indigestible food is nervous in character. We know that under these circumstances certain perversions of the gastric secretion occur, that abnormal fermentation-processes frequently develop, and that stagnation of the ingesta is thereby caused. A tendency to atony, as we have said, may also be congenital. I consider the so-called weak stomach that occurs in several members of a family to be hereditary. It has also been claimed that certain substances, as tobacco and coffee, may lead to atony of the stomach. These articles undoubtedly cause an increased secretion of gastric juice in some persons. It has never been positively demonstrated, however, that they produce primary atony. Atony is also occasionally seen in hysteric and neurasthenic subjects, and under these circumstances is probably of a nervous type. Those forms of atony that complicate certain diseases of the abdominal organs, particularly the peritoneum, can hardly be ranked as neuroses of the stomach proper. Atony of the stomach has also been seen to follow traumata, even injuries that did not affect the region of the stomach directly. In those cases in which the region of the stomach itself is directly injured we must always consider perigastric processes in the first place. Atony of the stomach, finally, has been seen as the result of gall-stone colic, and in this instance it is probably of nervous origin. Rosenheim[1] has very recently called attention to the development of atony as the result of the gastric crises of tabes.

I refer to the section on Motor Insufficiency for the diagnosis. In order to consider atony of the stomach a genuine gastric neurosis, we must, on the one hand, discover some primary etiologic factor, and, on the other, determine that no anatomic changes exist in the stomach, in particular that the secretion of gastric juice is not perverted. If secretion is perverted, we are more justified in concluding that atony is a result of this abnormality. The only way, of course, in which to decide whether the stomach is atonic and the secretion of gastric juice at the same time disturbed, is to analyze the stomach-contents. This analysis is also absolutely necessary in order to demonstrate the existence of atony. I consider atony, as a primary genuine neurosis, very rare.

Treatment must be directed, on the one hand, against the primary cause; on the other hand, against the atony itself. I refer to the section on Motor Insufficiency for the methods we possess for treating atony itself. The attempt should be made to raise the tone of the whole nervous system as far as possible, and to combat any neurasthenic and hysteric tendency that may exist.

Insufficiency and Incontinency of the Pylorus.—If a sufficient quantity of an effervescent powder is introduced into the stomach of a healthy subject, the organ becomes distended so that its outlines are distinctly palpable and may even become visible through the abdominal walls. The gas remains in the stomach for a few minutes and then slowly escapes, usually by belching, through the cardia. Normally the pylorus does not permit the passage of any gas at first, and does not relax for several minutes. The pyloric ring is also closed during digestion, and only relaxed from time to time in order to allow the escape of liquefied or pultaceous masses of food into the intestine. Normally, therefore, we see that the pylorus is continent, that its powers of closure are normal. In certain morbid conditions, however, we find the pylorus insufficient—that is, its powers of closure are abnormal, the sphincter of the pylorus is incapable of closing the pyloric orifice and of preventing the passage of ingesta from the stomach into the intestine.

Such a condition of insufficiency or incontinency of the pylorus may be due to organic changes, or may be due to some nervous disorder. The musculature of the pylorus may become destroyed by some ulcerative process, as, for instance, in carcinoma; or it may be replaced by unyielding cicatricial tissue. Stenosis of the duodenum or compression of the duodenum from without, or finally cicatricial stenosis of the duodenum, may all stretch the pyloric ring to an abnormal degree, and thus cause insufficiency. There are, however, a number of cases in which mechanical causes can be excluded, and in which the insufficiency of the pylorus is due to paresis of the motor nerves supplying the circular muscles. Ebstein was the first to call attention to this nervous form of pyloric insufficiency. This condition has been found in compression-myelitis and in hysteria. Cases in which this nervous form was found are exceedingly rare. I have been in the habit of inflating the stomach in nearly all the cases that I have treated; I do this as a routine measure in order to determine the position and the form of the organ; while I have frequently encountered insufficiency of the pylorus, I have never seen a case in which I felt justified in assuming that this insufficiency was purely nervous in character.

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[1] *Berlin. klin. Wochenschr.*, 1897, Nos. 11, 12.

The diagnosis of insufficiency of the pylorus is, above all, based on the observation that air forced into the stomach is not retained for any length of time, but immediately escapes through the pylorus. If an effervescent powder is swallowed, the stomach does not become inflated, but the intestine instead, and at the same time tympany is absent. Some authors object that this test is not valid, because a small quantity of gas would not cause distention of the stomach even though the pylorus were insufficient. The best way to avoid this error is, of course, to administer a large quantity of the effervescent mixture.

On the other hand, however, the stomach may become distended even though the pylorus is insufficient; for instance, if the insufficiency is due to an advanced degree of stenosis of the duodenum. Under these circumstances, however, it will rarely be difficult to diagnose stenosis of the duodenum or of the neighboring parts, for sooner or later the stomach will become dilated just as in stenosis of the pylorus. All other methods that have been recommended in the diagnosis of insufficiency of the pylorus are of subordinate importance, as, for instance, the salol test. If the latter is carried out early, appearance of the salicyluric acid reaction would indicate incontinency.

It is possible that the application of the spiral sound that Kuhn[1] has recently recommended may enable us to demonstrate the existence of pyloric insufficiency directly. So far, however, no investigations into this method have been made. The cessation of vomiting and the appearance of diarrhea are two symptoms that Ebstein considers particularly typical, but I think they will be found absent in many cases, and will be present only in those cases of insufficiency that are due to some organic lesion.

I do not consider the abnormally rapid passage of the ingesta from the stomach into the intestine as a valid criterion, although this symptom is frequently mentioned by different authors. I have seen many cases of organic insufficiency of the pylorus in which this symptom was absent. Another objection to it is that it also occurs in increased motility of the stomach.

If the existence of pyloric insufficiency has been determined, we must decide whether we are dealing with the nervous form or not. We are justified in diagnosing the latter only if we can positively exclude all organic changes, and if we can at the same time demonstrate that the patient is of a nervous disposition. A certain instability of the symptoms would also speak in favor of the nervous character of the disease; the pylorus, for instance, might close in a normal manner at certain times and at others be insufficient. If this temporary insufficiency occurs after certain injuries to the nervous system, the nervous character of the disease may almost be considered established. It is true, of course, that in certain organic diseases of the stomach the contractibility of the pyloric sphincter varies. The diagnosis of nervous insufficiency of the pylorus is at best a very difficult one.

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[1] *Arch. f. Verdauungskrankh.*, vol. iii.

Treatment, of course, can only be directed against the primary cause. Some authors have recommended massage, electricity, strychnin, and douches against the insufficiency *per se.*

Insufficiency of the Cardia, Regurgitation, and Rumination.—The circular muscles of the cardiac orifice may become insufficient in the same way as the corresponding fibers of the pyloric ring. Normally the contractions of the cardiac sphincter are less vigorous than those of the pyloric sphincter. This we have learned by studying the results of artificial inflation of the stomach by carbonic acid gas. We have seen that the gas always escapes upward at first, and that the spasm of the pylorus relaxes later than the spasm of the cardia. Air that is swallowed during eating and gases that are formed in the stomach can readily escape upward at once as soon as the cardia is insufficient; whereas if there is spasm of the cardia, the air and gas cannot, of course, escape. As long as nothing escapes upward but gas and air, the patients are not distressed, and are really not sick; as soon, however, as stomach-contents regurgitates upward, the patients suffer considerably. We speak of regurgitation if, after eating, some of the ingesta rise upward from the stomach and enter the mouth and have to be expectorated.

We speak of rumination if the ingesta that have risen into the mouth are immediately swallowed again. It is immaterial whether they are chewed again or not. Both processes, regurgitation and rumination, have this in common, that the food rises into the mouth: in the first condition, it is expectorated; in the second it is swallowed, and may or may not have been chewed again.

There is a great diversity of opinion in regard to the nature of regurgitation and rumination.

Some authors attribute this condition to permanent paresis and relaxation of the cardia; others assume temporary insufficiency of this orifice; still others attribute the disease to increased irritability of the vagus or of the dilator nerve of the cardia. This irritation, they state, may be central, peripheral, or reflex.

We have stated that in regurgitation, as distinguished from eructation, not only gas and air, but liquid and fluid particles of food also rise upward. At first this regurgitation is an involuntary act that calls for no effort on the part of the patient. It is not accompanied by nausea or vomiting. Some patients, however, can produce it artificially by contracting the abdominal muscles, in this way favoring the upward movement of the ingesta. The regurgitated material, of course, has a taste that differs according to the period of digestion during which regurgitation occurs. If regurgitation occurs very soon after the food is swallowed, it retains its original taste; later it acquires an acid or a bitter taste. As a rule, the patients are capable of suppressing the regurgitation of ingesta if they are energetically commanded to do so. This factor alone enables us to distinguish this form of regurgitation from the regurgitation seen in stenosis of the esophagus and in diverticula of the esophagus. The last-named diseases can, of course, also be differentiated from simple regurgitation by the subjective symptoms of the patient, by passing the sound, and by chemical analysis of the material raised, for the latter will reveal the absence of all changes that would have occurred had the food remained in the stomach for some time.

This nervous form of regurgitation is of subordinate importance as long as small amounts of food are raised. If regurgitation occurs frequently, however, and if abundant quantities of stomach-contents are raised, the general nutrition of the patient may suffer seriously. Nervous regurgitation, however, rarely becomes so severe.

The diagnosis is, as a rule, easy. In the first place, of course, we must exclude organic disease of the stomach or esophagus. Patients of this kind usually develop other hysteric or nervous symptoms. Some of them regurgitate food only when they know they are under observation, particularly if they are excited. It is usually possible to stop regurgitation by psychic suggestion. Treatment, therefore, must chiefly be directed toward influencing the patient to suppress regurgitation. In some cases regurgitation occurs more readily if the patients eat too fast, and if they do not thoroughly masticate and insalivate the food. Patients of this kind should be instructed to chew their food thoroughly and to eat slowly. Some clinicians advise swallowing pieces of ice, using massage, galvanization and faradization of the stomach, strychnin, and numerous other means and remedies. I am unable to say whether any of these measures help the patients; they may be tried in obstinate cases.

Whereas in regurgitation, as we have said, the food is expectorated, it is swallowed in rumination. In the latter condition temporary relaxation of the cardia is the primary factor. According to Singer, the stomach-contents is aspirated through the relaxed cardia. Mechanically, this aspiration can be explained by assuming that the air-pressure is decreased in the thoracic cavity during inspiration, particularly if the thorax is held in the inspiratory position while the glottis is closed.

I have mentioned that both regurgitation and rumination occur at the time of digestion. It seems probable, therefore, that rumination is connected in some way with certain secretory anomalies. This view, however, is wrong, for an analysis of the stomach-contents in different cases of rumination reveals altogether varying results. Even in the same individual the hydrochloric acidity of the stomach varied at different times. These variations alone show that whatever anomaly of secretion that might be present together with rumination could possibly be made responsible for this disease. As a matter of fact, secretory anomalies must be considered complications, or one of the symptoms, of the primary neurosis that causes rumination.

Very little can be said in regard to the etiology of rumination. The disease occurs in all classes. The majority of cases have so far been observed in men. Heredity, or, better, imitation, undoubtedly plays a certain rôle. Whenever rumination occurs both in parents and children I am very much inclined to believe that imitation is the primary factor in this coincidence. [Müller recently reported a family in which the father and two sons were ruminants. In the case of the father there was carcinoma of the walls of the stomach, involving the cardia, which was found to be abnormally wide; there was also cylindric dilatation of the esophagus.—Ed.] Occasional causes are emotional disturbances, psychic shock, rapid eating, and trauma of the region of the stomach. All these injuries, of course, cannot produce rumination in a perfectly healthy subject; a second factor must be present, namely—a certain nervous predisposition, and, as a matter of fact, rumination is nearly always found in hysterics, neurasthenics, epileptics, and idiots.

I have described the main symptom of this condition—namely, the entrance of fluid or solid stomach-contents into the mouth, and have also called attention to the fact that this material is swallowed, and not expectorated as in regurgitation. In general the food is not chewed, so that the name "rumination" is really a misnomer. Only in a small minority of cases, particularly in the insane, in idiots and in epileptics, do we find that the food is masticated before it is again swallowed.

We have mentioned above that the results of the chemical analysis of the stomach-contents are not uniform; in some instances we find hyperacidity, in others subacidity. Free hydrochloric acid may be completely absent, or the acidity of the stomach-contents may be normal. The motility of the stomach has been found normal in the great majority of cases. No changes in the general health or the general nutrition of the patient have, as a rule, been observed. It is a remarkable fact that most of the patients are able to suppress rumination, and that some sudden psychic shock may cause temporary disappearance of the phenomenon.

Rumination can readily be recognized, and it is impossible to confuse it with vomiting. As a rule, the diagnosis can be made from the description of the symptoms alone. It is still easier, of course, to recognize rumination if the patients can be observed.

Treatment is purely psychic. If the patients have acquired the bad habits of eating very rapidly and of not masticating thoroughly, these habits should be corrected. The patient should be systematically taught to suppress rumination. If the physician exercises a sufficient amount of psychic influence over his patient, and if the patient himself is willing, this usually succeeds. I need not mention that any secretory abnormality that may be present must be corrected. As a rule, however, we will find that these perversions of secretion are due to the general nervousness. This general condition, therefore, must be combated with all the methods that are at our disposal to raise the tone of the nervous system. Patients who have acquired the habit of rumination should be isolated as much as possible, as we know from experience that other patients may begin to ruminate if they see others afflicted in this way.

All other measures and remedies that have been recommended probably act indirectly—that is, psychically.

B. Secretory Neuroses of the Stomach.—The secretory function of the stomach is under the direct control of the nervous system. We know this by drawing analogies with other glandular organs and from our clinical experience. We know, for instance, that the secretion of gastric juice may be reflexly stimulated. This has been shown by the well-known experiment of Bidder and Schmidt,[1] who showed that in hungry dogs the mere sight of food causes secretion of gastric juice. I also refer to the observations of Richet,[2] who observed the secretion of gastric juice in a man with an impermeable esophagus and a gastric fistula as soon as he chewed food that was highly flavored, even though none of this food entered the stomach.

This and other experiments, however, did not determine what nerve was the true secretory nerve of the stomach until Pawlow and Schumowa[3] recently solved this problem. They performed esophagotomy in a dog, so that all food that was swallowed immediately passed out of the open end of the esophagus through the wound, and could not enter the stomach. At the same time they made a gastric fistula, through which they secured gastric contents for examination. As soon as the dog began to chew his food an abundant secretion of gastric juice occurred; if the vagus, however, was severed, this secretion did not occur. From these experiments they draw the conclusion that the reflex stimulus that causes secretion of gastric juice passes through the vagus.

Pawlow and Schumowa succeeded only once in stimulating the secretion of gastric juice by direct irritation of the vagus. The result of this experiment, however, must be considered ambiguous even though it was positive, for a small amount of food was still present in the stomach when the experiment was performed. In the second place, very much less gastric juice was poured out than when the animal chewed food. Schneyer[4] was the first to demonstrate positively that the pneumogastric nerve is the secretory nerve of the stomach.

It seems established, therefore, that the secretion of gastric juice can be influenced by nerve irritation. If we draw analogies with other glands, we may expect that this secretion may be both inhibited and stimulated by nervous impulses. Clinical observation seems to coincide with this view and strengthen this assumption. We frequently encounter cases in which the secretion of gastric juice is either increased or reduced, and in which these perversions in either direction can only be considered due to some nervous disorder; thus we speak, on the one hand, of nervous hyperacidity, of nervous gastrosuccorrhea; and, on the other hand, of nervous subacidity, or nervous hypochylia, and achylia gastrica. We are not justified, however, in considering every increase in the secretion of gastric juice as nervous, nor can we do so in the case of every reduction in gastric secretion. We have known for a long time that subacidity and anacidity may not be due to nervous disorders alone, and that in the majority of cases they are complications or a sequel of organic disease of the stomach. There is no reason *a priori* why the same should not apply to diseases in which the secretion of gastric juice is increased. We can speak only of secretory neurosis in those cases in which abnormal gastric secretion is exclusively due to some nervous trouble and originates from some reflex irritation or inhibition of the nerve of the stomach.

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[1] Bidder and Schmidt, "Die Verdauungssäfte und der Stoffwechsel," 1852.

[2] *Journal de l'anatomie et de la physiologie*, 1878.

[3] *Centralbl. f. Physiologie*, vol. iii., No. 6.

[4] *Zeitschr. f. klin. Med.*, vol. xxxii., p. 131.

Nervous Hyperacidity, Superacidity, Hyperchlorhydria.—I shall not enter into an exact description of the symptoms of this form, for I have done so in the section on Hyperchlorhydria. In that place I have also given my reasons for discussing hyperchlorhydria separately, and for not merely describing it here among the secretory neuroses. Hyperchlorhydria may be nervous in character, and frequently is, but this is not necessarily the case, for hyperchlorhydria is frequently seen under conditions that exclude a nervous origin. In many instances the primary cause of hyperchlorhydria remains uncertain, but this uncertainty does not justify us in declaring the disease in these instances to be a neurosis. In other cases we see hyperchlorhydria in connection with organic diseases of the stomach, as gastritis and ulcer. Hyperchlorhydria, as a matter of fact, is the rule in the last-named lesion. There is a great diversity of opinion in regard to the causal nexus existing between the two. At all events we are justified in saying that in ulcer hyperchlorhydria is not a neurosis in the true sense of the word.

Hyperchlorhydria is characterized by the secretion of an abnormally abundant and abnormally acid gastric juice during digestion. Both the total acidity and the amount of free hydrochloric acid are increased. It is commonly believed that the ferments of the stomach are also secreted in increased quantities, but this question has not yet been sufficiently investigated to warrant us in making any positive statements.

We can only speak of nervous hyperchlorhydria if we can determine that no organic disease of the stomach is present, and that the increased secretion of gastric juice is due to some nervous perversion. As a rule, patients of this kind will show some other general nervous symptoms if they are carefully examined. We most frequently find nervous hyperchlorhydria in erethistic types, in neurasthenics, and in subjects who have undergone excessive mental exertion. Hyperchlorhydria that is due to organic disease is characterized by regular and persistent increase of gastric juice, whereas in the nervous form the degree of gastric secretion fluctuates greatly. We will find the secretion of gastric juice normal at one time, increased at another, increased particularly if the patient sustains some violent psychic shock, undergoes mental overexertion, or exposes himself to some other influence that damages the nervous system. The nervous form of hyperchlorhydria, to judge from my personal experience, is more frequently encountered in the educated classes than among uneducated people; for instance, in physicians who have a very wearing practice, in lawyers, in savants, in merchants, in other words, in people who exert themselves mentally or lead exciting lives. The disease is most frequent in the middle years of life, rare in old people, and very rare in young subjects. The disease is less frequent in women, and occurs chiefly in hysterics.

I refer to the section on Hyperchlorhydria (page 299) for the symptoms and the diagnosis of this condition.

Hyperchlorhydria never represents a perversion of gastric secretion alone; as a rule, the sensibility of the stomach is increased at the same time, so that there also is hyperesthesia or gastralgia. Inversely, however, hyperesthesia or gastralgia alone can produce many of the symptoms that are considered typical for hyperchlorhydria. The only way in which to determine the presence of one or the other form is to aspirate the stomach-contents and to submit it to chemical analysis. Disturbances of the motor function of the stomach, particularly spasm of the pylorus and increased peristalsis, are also frequently seen together with hyperchlorhydria.

As a rule, the ingesta leave the stomach sooner than is normal in hyperchlorhydria, for the reason chiefly that gastric digestion proceeds very rapidly; occasionally, however, we may encounter a case of hyperchlorhydria in which there is apparently motor insufficiency, for the ingesta remain in the stomach an abnormally long time even though the secretion of gastric juice is increased. This retention of ingesta, however, is not due to atony, but, as a rule, to spasm of the pylorus.

The diagnosis of hyperchlorhydria is based exclusively on the results of the chemical analysis of the stomach-contents aspirated after a test-breakfast or a test-meal. The only way to determine whether we are dealing with the nervous form is to consider the etiology, the course of the disease, and the general condition of the patient. I consider frequent intermissions in the severity of the symptoms one of the most important criteria for the nervous form. If we see that the symptoms appear in attacks, remain altogether absent for weeks, and then suddenly reappear after some psychic shock or excitement, we may be reasonably certain of the diagnosis "nervous hyperchlorhydria." We should always be careful in rendering such a diagnosis, and should only make it if we can positively demonstrate the nervous origin of the trouble; in other words, we should only include such forms under this diagnosis in which no organic disease exists, and in which every other possible mode of origin can be positively excluded. The most important diagnostic features, as I have said, are the irregularity of the attacks and their dependence on psychic factors. Personally I do not consider this form of nervous hyperchlorhydria to be as frequent as some authors claim. It is true that in many cases it is difficult to determine the exact significance of hyperchlorhydria, but that is no reason to declare every hyperchlorhydria of uncertain origin a neurosis.

I will refrain from discussing the different methods of treatment and the various remedies that have been recommended for hyperchlorhydria ; I refer to previous sections for all this. In this place I will limit myself to discussing those methods that are directed against abnormal irritability of the stomach *per se.*

I have shown above that in the majority of cases the attacks originate, not from direct irritation of the secretory nerves of the stomach, but from some reflex irritation, and that the attacks usually occur in nervous and excitable subjects. It is not enough to treat each single attack, nor does it suffice to regulate the diet, however important this regulation may be in the general scheme of treatment, for we frequently witness attacks in persons who are exceedingly careful in what they eat. Precisely in these cases we see the patients who can eat the most indigestible food on one day are immediately afflicted with a violent attack of pain combined with hyperchlorhydria on some other day, even if they eat nothing but a very tender little piece of beefsteak. The character of the food in these instances is not responsible for the attack, but some nervous excitement ; the latter is capable, therefore, of producing an abnormal secretion of gastric juice, even though the diet is non-irritating. In a case of this kind the chief indication is to remove the primary cause—in other words, to raise the tone of the whole nervous system and to regulate the mode of life. In some persons a prolonged sojourn in the country, in others at the seashore, in a third person a cold-water treatment, may be the remedy. Very much will depend on the general strength of the patient, his customary mode of life, and the general status of the nervous system. It is quite impossible to formulate any general rules of treatment. We must remember chiefly that it is not sufficient to combat the symptom "hyperchlorhydria," but that the nervous predisposition must be corrected, the tone of the nervous system raised, and all causative factors removed.

Nervous Hypersecretion, Nervous Intermittent Gastrosuccorrhea, Nervous Continuous Secretion of Gastric Juice.—For the more important aspects of this form of secretory perversion I refer to the section in which I discuss it (see page 333). Gastrosuccorrhea, like hyperchlorhydria, is primarily a functional disorder ; the gastric mucosa in this perversion secretes large quantities of gastric juice without being irritated by food, in fact, when the stomach is empty ; consequently, abundant quantities of gastric juice are always present in the stomach, even though the organ has been quiescent for some time.

Gastrosuccorrhea may appear in an intermittent form, in attacks that occur at irregular intervals, or it may constitute a permanent condition that persists for some time. We can only speak of nervous gastrosuccorrhea in those cases in which the perversion of gastric secretion is due to some purely nervous cause. In general, gastrosuccorrhea is probably due to reflex irritation, and is rarely an independent primary neurosis of secretion.

Sahli[1] has reported a classic case of gastric crises in a tabetic subject in which large quantities of stomach-contents were vomited that contained no food-particles, but an abundant quantity of hydrochloric acid. This abnormal stomach-contents was raised even though the patient had eaten nothing for some time. In the interval between the attacks gastric secretion was quite normal. In this case, then, we are certainly justified in speaking of a secretory neurosis, and in particular of a form of nervous intermittent gastrosuccorrhea. Rossbach[1] has classified certain cases as nervous gastroxynsis, and includes in this category cases in which mental overexertion, excitement, or violent anger lead to the secretion of an abnormally large quantity of gastric juice. This occurs in subjects who are perfectly healthy otherwise, so that there can be no doubt that here, too, we are dealing with a nervous perversion or secretion.

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[1] *Correspondenzbl. f. Schweizer Aerzte*, 1885, vol. xv.

I do not think that nervous gastroxynsis can be regarded as a peculiar form of hyperchlorhydria and hypersecretion. Rossbach himself considers an abundant secretion of gastric juice produced by some nervous influence as the primary symptom in the disease he describes. The fact that headache, either in the beginning of the attack or later in the course of the disease, usually complicated the symptom-complex does not justify us in classifying this disease separately. The headache is merely a sequel of the hypersecretion, for it stops as soon as the stomach gets rid of its acid contents. In Rossbach's cases it has never been directly demonstrated that gastric juice is secreted when the stomach is empty, but the author claims expressly that attacks occasionally occur after prolonged periods of fasting. I am inclined to consider Rossbach's cases, as far as I can judge from the description, in part periodic attacks of hyperchlorhydria, in part cases of intermittent gastrosuccorrhea.

The intermittent form of hypersecretion is seen, above all, in very nervous subjects, in persons who undergo great mental exertion, and, occasionally, in hysterics and neurasthenics. This form is also, though less frequently, seen in gastric crises of tabes, occasionally in myelitis and in progressive paralysis. After the abuse of tobacco both hyperchlorhydria and gastrosuccorrhea have occasionally been observed.

Intermittent gastrosuccorrhea in the majority of cases has been shown to originate from some nervous disturbance. The continuous form, it appears, is not so frequently due to primary nervous disorders. The very fact that gastrosuccorrhea remains so uniform and persists for so long a time militates against the nervous nature of the disease. It is at present undecided whether or not some of these cases are secretory neuroses.

I shall not enter into a discussion of the character of this disease in this place, but refer to previous sections. I merely wish to emphasize one point. Some authors have objected that gastrosuccorrhea is really no pathologic symptom, because a small amount of gastric juice can usually be obtained from a perfectly normal stomach even though it contains no food. I do not, however, consider this objection valid.

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[1] *Deutsch. Arch. f. klin. Med.*, vol. xxxv.

I do not deny that a few cubic centimeters of gastric juice may occasionally be found in a healthy stomach that contains no food, but this may be due to a variety of causes. Saliva that is swallowed may produce some secretion of gastric juice. The amount of gastric juice excreted should, however, always be proportionate to the irritant that stimulates secretion. If we find from 3 to 10 c.c. of gastric juice, together with saliva, in a stomach that contains no food, we can certainly not speak of gastrosuccorrhea; but if we find from 200 to 300 c.c. of gastric juice in the stomach, there is certainly a disproportion between stimulation and secretion. If a perfectly healthy subject, after some mental overexertion or excitement, suddenly develops violent pain in the stomach, complains of headache, and vomits abundant quantities of acid gastric juice containing no food, then I think we may speak of abnormal secretion.

I refer to the section on Hypersecretion for the symptoms of gastrosuccorrhea and for the treatment of this disease, and refrain from describing these features in this place in order to avoid repetition. I will merely state, in regard to the nervous form that we are discussing, that we should endeavor above all to discover the primary cause of the abnormal stimulation of the gastric glands, and to remove it if possible. In many cases causal treatment cannot be carried out because we fail to discover the primary cause or because it is irremediable; in other cases in which the attacks are brought about by mental overexertion, we should instruct the patient to avoid all excesses of this kind and to take the necessary amount of rest—in other words, we should regulate his mode of life. Sea-bathing, cold-water cures, a sojourn in the mountains, etc., may all be advised for the purpose of raising the tone of the nervous system.

Depression of the Secretory Nerves of the Stomach, Nervous Subacidity and Anacidity, Nervous Achylia Gastrica.—We have seen that the secretion of gastric juice can be increased by nervous stimulation; in the same way it can be decreased. We speak of subacidity of the quantity of gastric juice secreted during digestion is abnormally small; we speak of anacidity if the secretion of gastric juice is altogether inhibited. Subacidity and anacidity are seen in a variety of organic diseases of the stomach, principally in carcinoma, severe gastritis, amyloid degeneration and atrophy of the gastric mucosa. We can only speak of nervous subacidity or anacidity if all organic diseases of the stomach can be excluded. If in addition positive signs of some nervous disorder can be discovered, the diagnosis may, of course, be considered established.

The terms "subacidity and anacidity" are really misnomers, for in the majority of cases we find that the secretion of hydrochloric acid alone is not decreased or inhibited, but that the secretion of pro-enzymes is also reduced or inhibited. Instead of using the term subacidity, we should, therefore, speak of "hyposecretion" or "hypochylia gastrica"; instead of anacidity, we should say "achylia gastrica."

In the majority of cases that have been studied the production of hydrochloric acid, or, better, of gastric juice, is merely decreased, not inhibited. The disease has so far been seen almost exclusively in hysteria, in neurasthenia, occasionally in certain spinal diseases, notably tabes. Nervous hypochylia and achylia can rarely be considered primary independent secretory neuroses.

Until recently the complete inhibition of gastric secretion was considered a sign of atrophy of the gastric glands. Einhorn[1] was the first to describe a case in which achylia gastrica persisted without change for five years, but in which at the expiration of this time the secretory functions of the gastric glands became re-established. Einhorn expressed the opinion at the time that this suppression of gastric secretion was due to some nervous disturbance.

In a number of cases of achylia gastrica small pieces of gastric mucosa have been torn off during lavage and have been found in the openings of the sound. These shreds have repeatedly been subjected to microscopic examination, and in many instances relatively normal glands have been discovered in them. But this finding does not by any means demonstrate that the loss of gastric function was necessarily of a nervous character. Only if we could demonstrate that in many cases of this kind the whole gastric mucosa is normal, or, at least, only slightly changed, could we speak of a truly nervous disorder.

Martius[2] has published a very excellent monograph on achylia gastrica. In his brochure he reports a number of cases of this perversion in which neurasthenic symptoms were present. The latter disappeared as soon as the general health of the patient improved, but achylia persisted nevertheless. This shows us that pronounced neurasthenic symptoms may be present together with achylia gastrica, but that this coincidence does not necessarily indicate that the two are related. Martius expresses the belief that there are two forms of achylia gastrica—the one caused by atrophy of the gastric mucosa, the other being either congenital or the result of some primary predisposition to the disease. The latter form is found chiefly in neurasthenic subjects.

The symptoms of nervous hyposecretion of the stomach are essentially the same as those of the organic form. Morbid symptoms, particularly stomach symptoms, may, however, be absent in both forms. As soon, however, as the motility of the stomach becomes impaired or reduced, or the intestine becomes irritated, severe symptoms appear. I have called attention to the fact (compare the section on Achylia Gastrica) that the functions of the stomach can be vicariously assumed by the intestine, provided the ingesta enter the intestine within a short time and before they undergo decomposition in the stomach. Von Noorden[3] has shown that the intestine is capable of assuming all the functions of the stomach whenever the stomach contains so little hydrochloric acid that it cannot perform the normal disassimilation of albumin. This explains why the peptic power of the stomach is frequently found to be completely or nearly completely lost, while gastric symptoms are, nevertheless, absent and the general health and nutrition of the patient are fairly well maintained. It is important to remember this fact, because it shows that hypochylia and achylia need not necessarily produce severe gastric symptoms. If we see such symptoms in achylia or hypochylia, we must always think of some complication.

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[1] *Arch. f. Verdauungskrankh.*, vol. i.

[2] Martius, *Achylia gastrica, ihre Ursachen und ihre Folgen. Mit einem anatomischen Beitrag von Prof. Lubarsch*, Leipsic and Vienna, 1897.

[3] *Zeitschr. f. klin. Med.*, vol. xvii.

The only way, therefore, to demonstrate the existence of a perversion of this kind is to investigate the chemism of the stomach. Free hydrochloric acid is always absent. If the production of hydrochloric acid is altogether inhibited, the total acidity of the stomach-contents is very slight, or it may not even be acid in reaction. The amount of ferments present usually corresponds to the amount of hydrochloric acid. If there is subacidity, pepsin and rennet are usually found, even though free hydrochloric acid may be absent; if there is anacidity, so-called achylia gastrica, the ferments are, as a rule, absent. As long as the motility of the stomach is intact the ingesta are propelled from the stomach within a normal time, even though digestion in the stomach is deficient or inhibited; as soon, however, as the motor powers of the stomach become reduced severe disturbances occur. The food undergoes abnormal fermentation and putrefaction just as in carcinoma of the pylorus with ectasy. Severe symptoms also appear if the duodenum becomes inflamed. Cases of the latter kind may present all the symptoms of intestinal catarrh and no symptoms of stomach-trouble—that is, the patients may complain of frequent attacks of diarrhea, of a feeling of tension, of gurgling, but of no real stomach symptoms. Oppler[1] has reported some cases of this kind.

It is well known that achylia gastrica also occurs in cases of severe anemia, but we cannot discuss this form here.

The diagnosis of subacidity or anacidity, or, better, of hyposecretion and achylia gastrica, is comparatively easy if the stomach-contents is analyzed; occasionally these conditions are recognized by chance.

It is much more difficult to decide whether or not we are dealing with the nervous form. If nervous, hysteric, or neurasthenic symptoms are present at the same time, and if the presence of carcinoma, toxic gastritis, amyloid or atrophy of the gastric mucosa, etc., can be excluded, the diagnosis of nervous hyposecretion and achylia gastrica is probable; at the same time, all these factors do not establish the diagnosis. I refer to the case that Martius reported, and that we have described above, in which the neurasthenic symptoms disappeared, whereas the condition of achylia persisted.

Every physician who has had opportunity to study many cases will agree with me that the origin of achylia gastrica is frequently obscure. I have repeatedly discovered achylia gastrica by chance, and without eliciting any clue as to the origin of the disease; it is possible that some of the patients were suffering from a nervous, possibly congenital, secretory perversion in the sense of Martius. The quantity of the ferments present is unimportant in rendering a decision, although many authors seem to attach particular importance to them, for in achylia gastrica, whatever its origin or character, the ferments are, as a rule, absent. In subacidity, of course, they are usually present. From a diagnostic point of view, the perversion of the production of gastric juice seems most important, particularly if it can be shown that the secretion of gastric juice is always reduced after some psychic shock and is normal at all other times. The only possible explanation for this change in the secretion of gastric juice must be some changing morbid stimulation of the nerves of secretion.

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[1] *Therap. Monatsh.*, March, 1896; and *Deutsch. med. Wochenschr.*, 1896, No. 32.

Treatment of various kinds must be instituted. In the first place, the primary cause of the trouble must be considered; any congenital secretory weakness or nervous predisposition must be combated. Unfortunately, this indication can be fulfilled only in a small minority of cases. Even though neurasthenia may be cured and the general health of the patient improved, achylia may persist (see the cases of Martius). In the second place, treatment must be directed toward maintaining the motility of the stomach, or reëstablishing it if it is lost, for as long as the motor powers of the stomach are good sufficient food is assimilated. All catarrhal conditions of the intestine should be prevented or treated if they supervene, for we know that catarrh of the intestine interferes with the compensatory function of the duodenum and leads to serious impairment of nutrition. The most important feature of the treatment, therefore, is careful regulation of the diet. The meals should not be large enough to cause distention of the stomach. The diet should be mixed. There is no reason for limiting the patient to an exclusively vegetable diet; as a matter of fact, I consider a vegetarian diet inappropriate because it favors the development of atony of the stomach. A portion of the albumin may be given in the form of artificial foods that are easily assimilated—for instance, nutrose. These artificial preparations are especially indicated if the appetite is reduced, and if diarrhea exists in addition to deficient hydrochloric acid secretion. As we cannot give meat in these cases, nutrose may be given as a partial substitute. It is a very simple matter to add 30 to 40 gm. of nutrose *pro die* to soup or milk.

The diet should be of an appropriate form, and should be as finely divided as possible. I refer to the section on Atrophy of the Gastric Mucosa, Achylia Gastrica, for all the details.

The best drug is hydrochloric acid; 10 to 20 drops may be given in a wineglassful of water several times during each meal. This method of administering hydrochloric acid can do no harm, at the same time we must remember that it is impossible to compensate the deficiency of hydrochloric acid in the stomach with doses of this size, or even with larger doses, particularly if the subacidity is very great or if there is anacidity. From a theoretic point of view pancreatin should be useful. This preparation, of course, digests albumin in an alkaline medium. I have employed pancreatin in a number of cases, but have failed to see any appreciable results.

Lavage of the stomach is necessary only in cases that are complicated by atony. Subacidity and anacidity *per se* do not call for this method of treatment.

Douches, electricity, and massage of the stomach may all be attempted for stimulating the secretion of gastric juice. Courses of mineral waters instituted for the purpose of stimulating gastric secretion should be carried out with great care. This applies particularly to saline waters, for drinking large quantities of water undoubtedly predisposes to atony.

(c) **Sensory Neuroses of the Stomach.**—Pain, and a variety of other disagreeable sensations, are encountered in many different diseases of the stomach. We can speak of a neurosis only in the cases in which these abnormal sensations are exclusively nervous in origin, and are in no wise due to organic changes.

Normally we do not recognize that we have a stomach ; we experience no other sensation in the region of the stomach when the organ is digesting than when it is at rest. The peristaltic movements of the stomach that cause expulsion of the ingesta produce no sensation whatever as long as the stomach is normal. The ingestion of warm or cold food or drink, of acid, sweet, or strong articles of food, makes no difference and produces no sensation in the stomach. In pathologic cases this is different, and as soon as the stomach is diseased we experience a variety of abnormal sensations. Sometimes these abnormal sensations in the stomach may be of purely nervous disorders. We call the latter nervous hyperesthesia or nervous pain in the stomach. It has not been established whether there is a sensory neurosis of the stomach in which the sensibility of the organ is reduced. As the stomach normally performs its functions without creating any sensation, we can hardly say whether there ever is anesthesia. Even if such a condition should exist, we could hardly expect characteristic symptoms. We could speak of an anesthesia of the stomach only in those cases in which the patient experienced no disagreeable sensation even when the organ is distended with carbonic acid gas, for we know that in normal subjects this procedure is always accompanied by some distress. Even in these instances, however, we should be hardly justified in speaking of anesthesia.

From a practical point of view only the irritative forms of the sensory neuroses of the stomach need be considered. These are, first, hyperesthesia, and second, gastralgia. The anomalies of sensation of hunger and satiation do not, properly speaking, belong to the sensory neuroses of the stomach.

Hyperesthesia.—The condition of hyperesthesia is a morbidly increased sensibility of the sensory nerves of the stomach. If the condition becomes severe, we speak of gastralgia. There is no difference in kind between the two, merely a difference in degree, gastralgia being the more advanced degree of abnormal irritation of the sensory nerves of the stomach. Numerous intermediary stages between gastralgia with violent attacks of pain in the region of the stomach and hyperesthesia can be arranged. In many cases we may be unable to decide which form we are dealing with. In pronounced cases hyperesthesia and gastralgia, however, can be differentiated without difficulty.

Hyperesthesia is characterized by a variety of abnormal sensations: a feeling of pressure, fulness, tension, burning, boring, etc., during digestion. As a rule, these sensations persist for some time after digestion. Abnormal sensations of this kind are encountered in the majority of organic diseases of the stomach. These, of course, we are not discussing in this place. The same abnormal sensations are occasionally seen as complications or symptoms of hysteria, neurasthenia, and a number of diseases of the central nervous system. In anemia and chlorosis we also occasionally encounter hyperesthesia. In the latter cases we are by no means justified, however, in declaring the hyperesthesia to be a true neurosis of the stomach, for we may only diagnose this condition if the stomach is intact, and in chlorosis and anemia, as we know, we frequently see perversions of gastric secretion, in particular hyperchlorhydria, so that the latter alone may be made responsible for the abnormal sensations, and may even produce attacks of cardialgia. In other instances small losses of follicular tissue may produce cardialgia.

Occasionally abnormal sensations that may even lead to nausea and vomiting are seen after direct injuries to the stomach; for instance, if the organ is habitually overloaded with indigestible food, or if salty, acid, or spiced food is eaten for a long time (Fleischer). In cases of this kind it is necessary to exclude every other possible disturbance, particularly any perversion of secretion, for only in those cases in which such perversions are absent can we speak of a neurosis of the stomach. Hyperesthesia is also frequently seen in persons who habitually eat very little. As soon as they eat more than usual, abnormal sensations are experienced in the stomach that ultimately produce nausea and vomiting.

Cases in which increased irritability of the sensory nerves appears only after fasting, and in which the introduction of food relieves these sensations, are less frequently encountered.

In certain patients hyperesthesia occurs only after the ingestion of certain articles of food, so that we may say that such persons suffer from a peculiar idiosyncrasy. To the same group belong the cases of so-called "weak stomach" that Fürbringer[1] has designated nervous dyspepsia. Patients of this kind usually enjoy excellent appetite, but complain of distress almost immediately after eating or drinking anything. This distress persists until the food leaves the stomach. The most injurious articles of diet are sugar, fat, and amylaceous material; they are almost certain to produce pain if introduced into an empty stomach. Of liquid foods, coffee is the worst in this respect. I must particularly emphasize the fact that in Fürbringer's cases the acidity and the motility of the stomach were both normal; there was merely hypersensitiveness of the sensory nerves of the stomach, or, better, of the nerves that transmit the sensation of pain, so that they acted abnormally to irritants that would not have caused pain in a healthy stomach.

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[1] *Berlin. klin. Wochenschr.*, 1893, No. 15, Balneologen Congress.

In other cases an increased secretion of gastric juice can be made responsible for hyperesthesia, so that the abnormal sensations are really secondary in character in this group.

Fürbringer emphasizes the fact that this form is particularly frequent in the higher classes. He considers luxurious living, mental overexertion, the excitement of business life, to be the most important etiologic factors, but emphasizes at the same time that the majority of these cases are not really neurasthenic, and that the stomach alone seems to be involved. According to Fürbringer, influenza also plays an important etiologic rôle.

We see, therefore, that the disease-picture of nervous hyperesthesia may vary. In some cases a variety of abnormal sensations—pressure, fulness, burning, etc.—appear after any kind of food; in other instances, only after certain articles of diet. As soon as the ingesta leave the stomach the distress is relieved, and pain rarely occurs when the stomach is empty. Occasionally the abnormal sensations may become so severe as to constitute real attacks of violent pain, so that the picture of gastralgia is simulated. Even vomiting may occur. Many of these patients, therefore, reduce the amount of food, because they believe that eating little will prevent the occurrence of the disagreeable sensations. In some of the patients true anorexia develops as a result.

Hydrochloric acid has occasionally been considered a cause of hyperesthesia, and some authors speak of a particular form of hyperesthesia for hydrochloric acid, and consider it an independent neurosis. Talma has described a syndrome that he attributes to hyperesthesia of the gastric mucosa to hydrochloric acid. Whether or not the symptom-complex he describes can be interpreted in this sense, I am unable to say. In another place I called attention to the fact that hyperchlorhydria could not be positively excluded in these cases. Löwenthal[1] has performed a number of experiments on healthy subjects and on persons with diseased stomachs, for the purpose of studying the sensations experienced after the administration of concentrated solutions of hydrochloric acid and of lactic acid. He failed to discover any hyperesthesia of the gastric mucosa even when the concentration of the solutions was very great. At the same time we must remember that advanced degrees of hyperchlorhydria do frequently produce pain. In these instances, however, the hyperchlorhydria is the primary factor, the pain the secondary one, so that in cases of hyperchlorhydria we cannot speak of hyperesthesia to hydrochloric acid in particular. Only in those cases in which abnormal sensations are experienced when the hydrochloric acidity is normal should we think of hyperesthesia to hydrochloric acid. Personally, I have never encountered a case that could be interpreted in this sense.

The diagnosis of nervous hyperesthesia of the stomach is based on the absence of organic disease of the stomach, on the absence of hyperchlorhydria or other disturbances of secretion, and on the absence of motor disturbances of the stomach. Other factors that indicate the nervous character of hyperesthesia in certain cases are the occurrence of general nervous or hysteric symptoms that need not necessarily be local and limited to the stomach. Sticker[1] has recently reported a number of cases of hysteric hyperesthesia of the stomach from my clinic. In all of these cases the stomach was sensitive to pressure, and this sensitiveness was strictly limited to the area corresponding to the stomach, so that by determining the extent of the sensitiveness to pressure the outline of the organ could be projected on the anterior abdominal wall even before the exact outline, position, and form of the stomach had been determined by other means. This area of sensitiveness extended further when the stomach was artificially inflated, but again remained strictly within the boundaries of the organ. It is true that in ulcer the whole region of the stomach is also painful to pressure, but in this disease we always find certain spots that are particularly sensitive.

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[1] *Berlin. klin. Wochenschr.*, 1892, Nos. 47–49.

In acute gastritis we may find analogous conditions to hysteric hyperesthesia, but this disease can, as a rule, be differentiated from nervous hyperesthesia without much difficulty. The course is different, and the analysis of the stomach-contents will reveal the well known disturbances of secretion, the presence of mucus, etc.

In the majority of cases of nervous hyperesthesia we find other nervous and hysteric symptoms that aid us in recognizing the nature of the disease. If we examine the patient with sufficient care we should not confound the disease with lesions of the abdominal muscles or diseases of the liver. The differential diagnosis between hyperesthesia and ulcer can usually be made from the analysis of the stomach-contents, for in the latter disease we always find hyperchlorhydria; in addition, the character of the pain is different, and the attacks of pain are more or less dependent on the quantity of the food.

If hyperesthesia is a secondary condition, one of the symptoms of hysteria, or of neurasthenia, the treatment should be directed against these general neuroses. If, on the other hand, we are dealing with an idiopathic neurosis of the stomach, we should proceed differently. Particularly in women and young girls do we find that such hyperesthesia causes the patients to eat less than they should, so that the general nutrition suffers greatly. In cases of this character we should endeavor to raise nutrition systematically. No general rules for doing this can be formulated. The best plan is to induce these patients to enter the hospital, for in clinics and hospitals this treatment can be instituted much better than at the home of the patient. I have repeatedly seen cases in which the patients refused to take any food on account of gastric hyperesthesia, and consequently became very much emaciated and exhausted. Patients of this kind should be in bed. The same applies to many of these patients who are not so emaciated or exhausted, for if they are instructed to go to bed and to avoid all physical and mental labor it is usually much easier to improve nutrition. If there is an advanced degree of anorexia, or if obstinate vomiting occurs as soon as food is introduced, nutritive enemata should be given for a time. Internally, small quantities of milk, if necessary with the addition of a little lime-water, tea, brandy, etc., should be administered, but only in teaspoonful doses. Chicken bouillon, veal bouillon, leguminous food, etc., may also be given, or nutrose may be added to milk or bouillon. Solid food should be given gradually and only in small quantities at first.

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[1] Sticker, "Beiträge zur Hysterie," *Zeitschr. f. klin. Med.*, vol. xxx.

Narcotics should be given only in exceptional cases. Galvanization of the stomach (the anode in the epigastrium, the cathode over the spinal column) has been recommended by a number of authors, and the anodyne properties of this method of treatment extolled. Rosenheim recommends silver nitrate, and claims that it acts as an anesthetic to the gastric mucosa. He gives 0.2–0.3 : 100.0 three times a day in the dose of a tablespoonful in a wineglass of water. He administers it in the morning before breakfast, and half an hour before dinner and supper.

Under certain circumstances sea-bathing, steel baths, and a sojourn in the mountains may be of benefit. Psychic treatment may also be indicated. In hysteric cases the personal influence of the physician usually accomplishes more than drugs.

Nervous Gastralgia, Cardialgia, Gastrodynia, Spasm of the Stomach.—Violent paroxysmal attacks of pain in the stomach are seen in a variety of diseases of the organ, particularly in ulcer. We are not dealing with these forms in this place, but only with those in which such attacks of pain occur independently of any organic change of the organ. We can only include such attacks of pain in the category of nervous gastralgia that are exclusively due to direct or reflex irritation of the sensory nerves of the stomach. The primary irritation may strike the peripheral endings of the sensory fibers of the vagus or the main stem of the nerve or its roots directly, or may be transmitted to the vagus by some reflex path.

Gastralgia appears in the form of paroxysms that occur at irregular intervals. Between the attacks there is usually no pain. The character of the pain is violent, boring, burning, tearing, or spasmodic and constricting. Occasionally it radiates anteriorly into the hypochondriac region and posteriorly toward the back and the spinal column. The attacks are independent of eating, and may occur when the stomach is full or when it is empty, at any time of the day or night. These attacks must be exceedingly painful, for, as a rule, the patients complain bitterly, and show from their facial expression alone how severely they suffer. The face is usually distorted with agony and cold perspiration breaks out on the forehead. The body is doubled up, and the region of the stomach pressed against some solid object, because pressure in the region of the stomach seems to relieve the pain. It is a peculiar fact that light pressure is experienced disagreeably, whereas strong pressure usually relieves the pain.

Other symptoms that appear are belching, hiccough, frequent yawning, nausea, occasionally bulimia, violent headache, and general depression,—in fact, the patient may go into collapse. The pulse is usually accelerated, small, occasionally intermittent, and in rare instances very much retarded.

The attacks usually appear suddenly and unexpectedly. In some instances they are preceded by mild prodromal symptoms, a feeling of unrest in the stomach, slight pain, nausea, and belching. As a rule, however, as we have said, the attacks occur suddenly and without demonstrable cause. Occasionally they follow some psychic shock, or appear regularly at the time of menstruation or immediately before. In general they recur at irregular intervals. Only in malaria do they occur with more or less regularity.

The duration of an attack of gastralgia may vary greatly. It may last a quarter of an hour or somewhat longer, or may persist for several hours. The frequency of the attacks also varies. Occasionally several attacks occur in one day; sometimes an attack occurs every other day, or at intervals of weeks and months. Sometimes gastralgic attacks alternate with other forms of neuralgia.

The patients usually recover rapidly from these attacks, provided they do not last extremely long and do not recur very frequently. The patients feel relatively well in the intervals between attacks; unless, of course, they are afflicted with the secondary form of gastralgia, and not the idiopathic primary form.

Cases of gastralgia that are exclusively due to increased irritability, that is to say, to a primary functional disturbance of the sensory nerve-endings, are rare. Cases of ulcer in which these nerve-endings are directly irritated cannot, of course, be included among the genuine neuroses. Gastralgia may, of course, also appear as a secondary symptom in certain purely functional disorders, as gastrorrhea and hyperchlorhydria. The attacks are due to the increased peristalsis of the stomach that in its turn is induced by the perverted or rather increased secretion of gastric juice, and at the same time causes abnormal, painful sensations in the stomach. The same applies to the gastralgic symptoms seen in perigastritis and in cases in which peritonitic adhesions have formed. The latter are distinguished from purely nervous cardialgia by the regularity of the attacks. Cardialgia is also occasionally seen in chlorotic and anemic subjects. Some authors, therefore, consider chlorosis and anemia as diseases that may lead to gastralgia. To judge from my personal experience, pure nervous cardialgia is rare in these diseases, and simple hyperesthesia is more frequent than violent cardialgia. In many of these cases, moreover, pain is secondary to hyperchlorhydria, and in still other instances some other disease, as an ulcer, a hemorrhagic erosion, etc., may be the primary cause. In any case of chlorosis and anemia that is complicated by violent attacks of cardialgia the diagnosis "nervous cardialgia" should be made only after repeated examinations, particularly of the gastric secretion.

Cardialgia has also occasionally been seen in arthritis urica and in malaria. In the latter disease cardialgia has been known to occur at the same time as the typical malarial attack; or, again, malaria may become manifest in the form of gastralgia just as it appears occasionally in the form of other neuralgias. In cases of this character the etiology of the disease can be cleared up by studying the effects of the administration of quinin. [Quinin is useful in many non-malarial cases of gastralgia.—Ed.]

Cases in which pressure on the vagus and sympathetic can be made responsible for gastralgia are very rare. Isolated instances of tumors have been reported that compressed the vagus or the sympathetic, and were possibly the cause of attacks of gastralgia. Diseases of the brain and spinal cord,—for instance, myelitis, multiple sclerosis, and exophthalmic goiter—have more frequently been known to produce gastralgia. Tabes is the most prolific cause of this disease, and the so-called gastric crises are really attacks of gastralgia. In the great majority of cases, however, gastralgia, like other neuralgias, is due to some hysteric or neurasthenic predisposition. Sexual overirritation and sexual diseases in general are among the chief causes of the disease. Peyer[1] has called attention to the fact that spasms of the stomach occur quite frequently as the result of lesions of the male genitals. They occur particularly when the stomach is empty. Diseases of the female sexual organs still more frequently lead to neuralgia of the stomach; in many women and girls cardialgic attacks occur regularly at the time of menstruation or immediately before it; in some instances they seem to take the place of menstruation.

Sexual disorders *per se* are not the sole cause of the cardialgic attacks; another factor—namely, abnormal irritability, must also be present. If this were not the case, attacks of gastralgia would occur in all cases of sexual disorders, whereas, as a matter of fact, they occur only in a small percentage.

Fliess[2] has attempted to demonstrate that the nervous gastric pain that occurs together with nervous dysmenorrhea is, as a rule, due to irritation of a distinct area within the nose. According to this author, neuralgia of the stomach is due to irritation of an area that is situated in the anterior third of the left middle turbinate. If the turbinates are removed in these cases, the attacks of gastralgia stop; or if this portion of the turbinates is touched with a 20 per cent. solution of cocain during the attack of gastralgia, the pain in the stomach disappears within five to eight minutes, and remains absent as long as the local action of the cocain persists. Fliess emphasizes particularly, however, that the gastric crises of tabes and the stomach pain of hysteria and chlorosis are not amenable to nasal treatment.

Diseases of the liver, the kidneys, the spleen, the bladder, and the pancreas, may all occasionally lead to reflex gastralgia.

Gastralgia is more frequent in women and young girls than in men, for the nervous system of women is, as a rule, more high-strung, and women are more prone to disorders of the sexual organs. I repeat that the last-named disorders alone produce neuralgia only in persons who are particularly predisposed. The same applies to men whose nervous system has lost tone, and in these subjects gastralgia is also quite frequent. The disease occurs in all classes; is most frequent between fifteen and forty years, less frequent in early youth. The disposition to all forms of neuralgia decreases with advancing years, and this also applies to gastralgia.

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[1] Peyer, "Ueber Magenaffectionen by männlichen Genitalleiden," *Samml. klin. Vortäge*, No. 356.

[2] Fliess, "Magenschmerz und Dysmenorrhoe in neuem Zusammenhange," *Wien. klin. Rundschau*, 1895.

The prognosis is favorable, for the attacks *per se*, however violent they may be, and even if they lead to severe symptoms of collapse, rarely endanger life. The prognosis depends on the primary cause; if this can be removed, the attacks of gastralgia will stop.

The diagnosis is rarely difficult. The sudden onset of the symptoms, the violent spasmodic pain in the region of the stomach, belching, nausea, headache, even vomiting, the occurrence of the attacks at irregular intervals, the fact that they are independent of eating, frequently occur after some emotional shock or mental overexertion, and stop suddenly, all aid us in rendering the diagnosis "gastralgia." Even if we know, however, that the symptom-complex is due to gastralgia, it remains to determine whether we are dealing with the nervous idiopathic form or with the secondary form. None of the symptoms we have enumerated can be considered pathognomonic, but the general syndrome is more or less typical. Some authors are inclined to attach particular importance to a certain isolated symptom, but I do not consider this justified in any case. Some clinicians attach particular importance to the arrest of pain by deep pressure over the stomach, and consider this a diagnostic criterion. This symptom is frequently observed, but it does not by any means demonstrate that the disease is gastralgia; nor is it constantly present in this disease. It is also true that nervous pain in the stomach is frequently mitigated by the application of galvanization, but this symptom, too, is inconstant and proves nothing.

If we encounter the above symptom-complex, we may know that we are dealing with an attack of gastralgia, but we know nothing in regard to its origin. If we see the patients during the first attack, it is frequently impossible to determine its cause; the only way to arrive at a decision and to exclude organic diseases of the stomach is to subject the patient to repeated examinations, and to aspirate the stomach-contents on different occasions. The differential diagnosis between ulcer and hyperchlorhydria on the one hand, and gastralgia on the other, is, as a rule, particularly difficult. I refer to the section on Ulcer for the diagnosis of this lesion. In pronounced cases it is an easy matter to differentiate ulcer from gastralgia, for in ulcer the attacks of pain occur with greater regularity and at the height of digestion, and, provided no complications exist, never when the stomach is empty. The attacks, moreover, are directly dependent on the quality and the quantity of the food. Finally, hyperchlorhydria is almost always present in ulcer, and I consider this one of the most valuable criteria. Wherever I find hyperchlorhydria I know at once that the attacks of pain are not a primary condition, but are secondary to some other disease of the stomach.

It remains to be decided whether or not hyperchlorhydria is genuine, or nervous, or whether it is merely a symptom of ulcer. I refer to the section on Hyperchlorhydria for this matter. All we care for in this case is to demonstrate the presence of hyperchlorhydria, for this at once excludes primary gastralgia or any primary sensory neurosis of the stomach. Rheumatism and myalgia of the abdominal muscles are probably never easily confounded with gastralgia. In these diseases the pain is constant, never occurs in paroxysms, is not limited to the region of the stomach, but corresponds to the course of the muscles, and is relieved when these are relaxed. In general some direct cause for the condition can be discovered.

Intercostal neuralgia can be taken for gastralgia only on very superficial examination. The pain can usually be traced along the course of the intercostal nerves as far as the spinal cord, and distinct painful pressure-points can usually be found.

It is occasionally more difficult to make a differential diagnosis between gall-stone colic and gastralgia. If icterus, acute swelling of the liver, and enlargement of the gall-bladder can be determined, the diagnosis of gall-stone colic is, of course, easy. But even without these typical symptoms we frequently succeed in making the diagnosis. In gall-stone colic the pain is felt chiefly over the liver and gall-bladder, whereas in nervous gastralgia the whole region of the stomach is, as a rule, painful. Deep pressure over the region of the stomach frequently relieves the pain in gastralgia, whereas deep pressure over the region of the liver always elicits violent pain in gall-stone colic. Typical attacks of gastralgia are occasionally seen in the intervals between attacks of gall-stone colic. The exact origin of this form of gastralgia is not established.

Kidney colic should not be confounded with gastralgia, for here the pain is felt in the region of the kidney and radiates into the ureter and the bladder of the affected side. If concretions and coagula of blood are found in the urine, the diagnosis may be considered established; but even if this is not the case, the differential diagnosis is usually easy.

After the diagnosis gastralgia has once been made, the cause of the trouble must be discovered. The only way to determine whether or not gastralgia is due to some organic disease of the stomach or is a sequel of such disease, is to examine the stomach repeatedly, in particular to analyze the stomach-contents frequently. The latter method is the only way to determine whether gastralgia is due to some secretory perversion. If the nervous character of gastralgia is determined, the primary cause should be looked for. Occasionally this is an easy matter—for instance, in nervous individuals, in whom the attacks occur whenever they sustain some psychic shock. In other instances it may be difficult, and may be possible only if the whole body is repeatedly examined.

The main difficulty in rendering a diagnosis is the fact that gastralgia is frequently not a pure sensory neurosis, but is often complicated with secretory and motor perversions. In treating these cases it is very important to know whether the primary factor is a spasm that secondarily leads to irritation of the sensory nerves, stasis of stomach-contents, and, as a result, increased gastric secretion, or whether the primary factor is a secretory neurosis that secondarily produces spasm and in this way pain; for treatment should always be directed against the primary disorder, not its sequelæ.

Treatment should be instituted in two directions: in the first place, it should attack the primary cause, and in this way prevent the recurrence of attacks; in the second place, it should combat the attacks themselves, attempt to mitigate them, and to stop them as rapidly as possible.

If the disease is primarily due to chlorosis and anemia, preparations of iron, a rest-cure, etc., are indicated. Violent attacks of nervous cardialgia are, however, as we have seen, rarely due to chlorosis or anemia. In these diseases hyperesthesia of the stomach and mild attacks of pain are more frequent. In all these cases we should, above all, carefully determine whether or not the attacks of cardialgia are possibly due to ulcer, to hyperchlorhydria, or to some other cause.

If sexual overirritation or disorders of the sexual organs are the primary cause of the disease, treatment should be instituted against these troubles. The same applies to those cases that follow gout, malaria, or tabes. Here, too, treatment should be instituted against the primary disease. If gastralgia is due to an hysteric or neurasthenic tendency the treatment should be directed toward raising the tone of the nervous system. Whenever we can find a starting-point for the attacks, treatment should be directed against it.

In combating the attack itself narcotics should be employed. If the attacks of cardialgia are very severe, morphin should be given subcutaneously, provided there is no idiosyncrasy that prohibits the exhibition of this drug. Other narcotics may also be employed. Warm compresses and cataplasms are useful in any case; less useful are sinapisms. Lavage of the stomach is only indicated where the ingesta are still present in the organ when the attack begins.

Galvanization of the stomach (the anode over the painful point or intraventricularly, the cathode over the spinal column) frequently relieves the pain in a short time. The faradic current is less effective.

Chloroform water given internally, or irrigation of the stomach with chloroform water or with carbonated water, has been found useful in many cases.

In hysteric subjects numerous other remedies may, of course, be employed. Here, as in most other nervous disorders, suggestion plays an important rôle.

ANOMALIES OF THE SENSATIONS OF HUNGER AND SATIATION.

Anomalies of the sensations of hunger and satiation, and of the appetite, may be briefly discussed in connection with the other disturbances of sensation: they are, first, exaggerated appetite, so-called bulimia or hyperorexia; and, second, lack of appetite, loss of the sensation of satiation, so-called anorexia or akoria.

Bulimia, Hyperorexia, Canine Hunger.—Bulimia is a morbid increase of the sensation of hunger that usually occurs in attacks either as an independent neurosis or as the result of some other disease. It is usually assumed that the center for the sensation of hunger is located in the medulla, and that this center is irritated as soon as the blood becomes impoverished to a certain degree. The sensation of hunger, therefore, must be considered a general sensation that does not originate in the stomach, but is projected into the stomach. Other authors postulate the existence of specific nerves of hunger in the stomach, and claim that irritation of these nerves causes the sensation of hunger. The existence of such nerves, however, has so far not been demonstrated.

There can be no doubt that the stomach is in some way concerned in producing the feeling of hunger, for this sensation parallels the secretion of gastric juice and the motility of the stomach in certain diseases of the organ. The amount of food present in the stomach also influences the sensation of hunger to a certain extent. Patients with hyperacidity and hypermotility of the stomach frequently complain of hunger. After gastro-enterostomy, patients who had been eating very little before the operation begin to complain of frequent hunger. Recently, for instance, I studied a case of cicatricial stenosis of the pylorus with ectasy. In this patient the appetite and the sensation of hunger were completely lost. A gastro-enterostomy was performed, and it was found that the ingesta left the stomach in a very short time. The patient at the same time began to complain of being hungry at frequent intervals, and usually very soon after he had eaten something. Observations of this kind teach us that the stomach, too, is concerned in producing the sensation of hunger, and is capable of influencing the hunger center.

Inversely, we frequently see that advanced impoverishment of the blood fails to produce the sensation of hunger, or that the sensation of hunger is suddenly suppressed ; for instance, by the aspect of something disgusting, by sudden fright, some violent emotional disturbance, etc.

We see, therefore, that the sensation of hunger may be stimulated and inhibited in different ways, both directly and indirectly. The same applies to the pathologic increase or inhibition of this sensation, for various causes may lead to these perversions.

Bulimia is occasionally seen in certain psychoses, in various tumors of the brain, in a variety of focal diseases, also in Basedow's disease, in epilepsy, hysteria, and neurasthenia. In these cases the hunger center itself is in all probability directly subjected to some abnormal irritation.

Bulimia is less frequently seen in diseases of the stomach. Here it occasionally occurs in those forms in which the secretion of gastric juice is increased and the motility is exaggerated. Hypersecretion most frequently leads to bulimia ; next in importance is hyperchlorhydria. In the latter disease the abnormal irritation undoubtedly originates in the nerves of the stomach, for here bulimia can certainly not be due to an impoverishment of the blood or to abnormal irritation of the hunger center. Bulimia may also originate from the intestine. It has been seen in diarrhea and in tapeworm. The increased appetite that we witness in convalescents, particularly in patients who are recovering from some severe febrile disease or from severe hemorrhage, or from childbirth, is not pathologic; it is merely a physiologic symptom, and is an expression of the demands of the body for nourishment to replace the loss sustained during the course of the disease.

In diabetes mellitus bulimia is occasionally, though not frequently, seen. In many of these cases the appetite is merely increased as a direct result of the increased metabolism. In other cases we see sudden attacks of canine hunger even after abundant meals. Possibly the sugar circulating in the blood irritates the hunger center. The fact that the morbid increase of the appetite is sometimes relieved as soon as mellituria is stopped by an appropriate diet favors this view.

Bulimia is more frequent in women than in men, and is most common between twenty and forty years.

The symptoms are so characteristic that we can never be in doubt in regard to the diagnosis. The exact point at which bulimia begins, however, is hard to determine. In pronounced cases canine hunger appears suddenly, occasionally immediately after a large meal; as a rule, other disagreeable sensations, even pain and faintness, may be complained of, so that the patient urgently calls for food. In some instances the attacks occur during the night, so that the patient is awakened by a feeling of hunger. Often a few morsels of food, a swallow of wine, or a little milk will relieve this abnormal sensation; in other instances large quantities of food or fluid have to be taken. If the patient is unable to satisfy his appetite at once, a variety of other symptoms are apt to appear—namely, faintness, palpitation, a feeling of fright and anxiety, etc.

In some patients these attacks occur with great frequency, so that the patients suffer from bulimia every few hours, even though they eat a great deal. In other cases the attacks occur at long intervals or alternate with anorexia. Bulimia may be very severe or very slight. Before we can speak of bulimia we must determine that the attacks occur suddenly, and that the patients express an urgent demand for food, and state imperatively that they must have something to eat.

The prognosis and the course of the disease naturally depend on the primary cause. If bulimia is an idiopathic independent neurosis, it is essentially impossible to make a definite prognosis. In general it may be said that the more frequent the attacks and the more violent, the less favorable the prognosis. There is always danger of gastritis, atony, or ectasy of the stomach as a result of the ingestion of the large quantities of food that the patients take. In the secondary form the course and the prognosis naturally depend on the primary disease. If we succeed in curing the latter, bulimia will also disappear. In some instances, however, bulimia has been known to persist even after the primary trouble was removed.

If violent attacks of canine hunger suddenly appear immediately or very soon after a large meal, the diagnosis is established. We must distinguish between polyphagia and bulimia. Polyphagia is the demand for large quantities of food, and the patients only feel satisfied if they eat large meals. These cases do not feel hungry until the food is digested. The distinction between bulimia and akoria may be more difficult; akoria is loss of the feeling of satiation. Patients with akoria never feel satisfied even though they eat large quantities of food. Patients with bulimia suffer from paroxysmal attacks of irresistible hunger and urgently call for food. Bulimia in these patients may disappear even after eating a very small amount. Patients with akoria, on the other hand, do not suffer from an abnormal sensation of hunger; as a matter of fact, may suffer from anorexia; they are abnormal in this respect, however, that after eating they do not experience the feeling of satiation that is considered normal.

Treatment should be directed against the primary disease in all those cases where bulimia is a secondary symptom (and this may be considered the rule). In many cases, however, it is impossible to combat the primary disease. If bulimia is one of the symptoms of hysteria, of neurasthenia, we must attempt to treat these diseases and to strengthen the nerves. If the attacks are started by some psychic shock, we should carefully avoid such injury, and at the same time attempt to raise the tone of the nervous system. I need not enter into the details of this treatment.

If the abnormal sensations we have described are due to increased secretion and motility of the stomach, these abnormal conditions should be relieved. The drugs that have been used to reduce the direct or reflex irritability of the hunger center are the bromids, opium, and arsenic. Some authors advise cocain. Patients who are afflicted with morbid hunger should always carry food with them.

Akoria.—Bulimia in some respects resembles the condition that we call akoria. Akoria means loss of the feeling of satiation. Patients with akoria never feel satisfied, even after eating a large meal. The sensation of hunger, the appetite, need not necessarily be increased; as a matter of fact, akoria may be present and the patients suffer from anorexia,—that is, loss of appetite.

I have mentioned the fact that polyphagia and akoria are by no means identical, for in the first condition the desire for eating is increased, whereas in akoria this is not the case. In polyphagia the feeling of satiation occurs but late,—that is, only after large quantities of food have been eaten.

Akoria is seen in hysteria, in neurasthenia, in certain psychoses, occasionally in diabetes, and, in rare instances, in ectasy of the stomach. The feeling of satiation fluctuates normally; the amount of food necessary to produce this sensation varies in each individual, and differs in the same individual according to the time of day. Relief of the sensation of hunger and the sensation of satiation are by no means identical. Normally, we speak of being satisfied only after a certain amount of food has been introduced; we may not be hungry even though the stomach is empty, but we may, nevertheless, not feel satiated. Loss of the feeling of satiation is by no means identical with hunger; I do not, therefore, think that we are justified in considering akoria simply an increased irritability of the hunger center. Even if the sensation of hunger is relieved, the patients, nevertheless, do not feel completely satisfied. Most people do not regulate the amount of food they eat according to the degree of hunger they experience, but according to their appetite. *A priori*, therefore, the assumption that loss of the sensation of satiation is due to reduced sensibility of the stomach seems probable. If this be the case, the patients should not experience the same disagreeable sensations as normal subjects when the stomach is inflated with air or carbonic acid, or distended with water. So far, however, this point has not been established. There is no typical disease-picture of this condition. The only symptom that we can consider typical is that the patients never feel satisfied, not even after an abundant meal. They cannot, therefore, regulate the amount of food they eat according to their subjective sensations, but must calculate the amount of food necessary from their experience before they became afflicted with the neurosis. Other neurasthenic or hysteric symptoms may be seen together with akoria.

The prognosis is dependent on the primary disease.

In treating these cases we should establish, above all, whether or not the stomach is abnormally distended. If this is the case, the dietary regulations and the physical methods of treatment that we have discussed in the sections on Atony and Ectasy should be employed. At all events, the primary disorder, hysteric or neurasthenic tendencies, etc., should be combated.

Anorexia Nervosa.—Anorexia is decrease or complete loss of appetite, that may become so pronounced that aversion and even disgust for food develop. In this place we cannot, of course, discuss all those forms of anorexia that are due to organic diseases of the stomach, nor can we discuss those cases in which the patients do not eat because they know they will suffer distress as soon as food enters the stomach. Patients of the latter class do not suffer from loss of appetite and do not refrain from eating because the appetite is reduced, but merely because they are afraid of the pain. In many instances of this character, however, prolonged abstinence may secondarily lead to anorexia. In this section we will limit ourselves to discussing those forms of anorexia that are purely nervous in character and develop independently of any organic disease of the stomach.

Anorexia nervosa occasionally appears in perfectly healthy subjects after some violent sudden psychic shock or after some depressing emotional disturbance. Bad news may cause loss of appetite in a perfectly healthy person; some disgusting sight, the taste of some disgusting article of food, a disagreeable odor, may cause aversion to any kind of food.

In other cases anorexia is not a transitory state, but a persistent perversion. The chronic form is seen in hysteria, in neurasthenia, and in certain psychoses. It is more frequent in women than in men, and usually originates on the basis of anemia and chlorosis. Anorexia is also seen in morphin-fiends and in excessive smokers.

Mild degrees of loss of appetite are frequently seen. There are numerous transition-forms from these mild degrees to the most severe ones that are characterized by aversion to any kind of food and attacks of vomiting that occur at the mere sight and smell of food. In many cases the appetite is merely reduced, not lost, so that the patients still eat a little; as soon, however, as they swallow a few morsels a feeling of aversion is experienced. In still other cases the patients have no appetite whatever, but are, nevertheless, able to eat a certain amount of food. Anorexia of this kind may persist for a long time. I recently had under my care a lady who was afflicted with anorexia, and who had reduced the amount of food she ate to a very small quantity. When I saw her for the first time the total quantity of food that she could be induced to swallow in the twenty-four hours was two to four teaspoonfuls of bouillon. The patient was so weak and debilitated that she was unable to sit up in bed without assistance. All hope of recovery had been given up. Her weight was reduced to sixty-four pounds. Very slowly, I succeeded in inducing the patient to take a little more food, and for a long time she had to be fed by rectum in addition. The patient ultimately recovered, although the process was a tedious one. Ten months later she was dismissed, weighing one hundred and thirty-four pounds. This is an example of advanced anorexia that nearly led to death.

In order to diagnose anorexia we must be able to exclude organic disease of any part, not only of the stomach. To do this we must, of course, subject all the organs of the body to a careful examination and carefully analyze the functions of the stomach. In many instances the patients will have to be examined several times in order to determine whether or not we are dealing with the purely nervous form, or whether anorexia is due to some hidden disease, for instance, incipient tuberculosis, etc.

The only way to treat severe and obstinate cases is to place them in a sanitarium. Cases that are addicted to the morphin-habit or the use of tobacco should, of course, be cured of these habits. If hysteria or neurasthenia is the primary cause of the trouble, the state should be attacked directly. If there are anemia and chlorosis a course of iron should be begun at once. In mild cases of anorexia a change of location, sometimes a trip to the seashore, will improve the appetite. In severe cases in which the general nutrition has suffered greatly a systematic cure must be instituted in some sanitarium. Bitters and stomachics have been recommended by many clinicians; also lavage of the stomach. All these measures may be tried, although I personally have never seen good results from them in severe cases. If the patient refuses all food, if he cannot be induced to eat by suggestion, nutritive enemata should be given or the patient should be fed through the stomach-sound. If this is done, very small quantities of food should be introduced at first and the dose gradually increased, chiefly because the stomach is unable to master large quantities of food after having been accustomed to a slender diet for so long a time. If too much food is given, the patient will suffer distress and may have to vomit, etc. Fleiner recommends the introduction of water that is heated to blood-temperature into the stomach, in order to distend the organ and to stretch the contracted walls of the organ. He then allows the wash-water to run out and slowly introduces a certain amount of food. Personally I have never been forced to employ this method, and have always succeeded without it; at the same time I consider it worth a trial. Fleiner recommends oatmeal with milk in particular. Artificial foods may be added to the milk or to meat broth. Personally I am most in favor of nutrose. Above all, it should be remembered that the amount of food should be increased very gradually. Patients of this kind should, of course, remain in bed; as a matter of fact, in severe cases they are unable to be out of bed. If the treatment is instituted in this systematic way the patients, as a rule, begin to eat spontaneously after a short time. The fact that they can retain some of the food that is introduced undoubtedly acts favorably in a psychic sense. Suggestion, as in the case of all other neuroses, is also of paramount importance in these cases.

In the preceding paragraphs I have described the different kinds of nervous functional perversions of the stomach that involve the secretory, the sensory, and the motor spheres. In the introductory remarks and in describing these different forms I called attention to the fact that in the majority of cases of nervous disorders of the stomach combinations of these different perversions occur, and that we are rarely dealing with a perversion of secretion, of sensibility, of motility alone; in other words, we usually encounter combined gastroneuroses. In many of these cases a connection between these different morbid symptoms can be shown to exist; in some cases this is impossible. Combined neuroses of this kind are called nervous dyspepsia or neurasthenia gastrica.

NERVOUS DYSPEPSIA (NEURASTHENIA GASTRICA).

Leube originally gave the name "nervous dyspepsia" to those nervous disorders of the stomach in which digestion was normal in regard to time and chemism, but in which the patients complained of a variety of distressing symptoms during the process of digestion, so that the assumption was justified that the nervous system was in some way involved. All these forms would, therefore, primarily be neuroses of sensibility. Leube's observations, however, do not demonstrate positively that the secretory functions of the stomach were normal in all these cases. I have emphasized the fact that the stomach may be empty seven hours after a test-meal and secretory perversions still exist; on the other hand, in Leube's cases the symptoms were not limited to the sensory sphere. Now-a-days we understand under the name of "nervous dyspepsia" a combined disease-picture; the syndrome may, of course, vary, but we always encounter sensory disturbances, distress of various kinds, pressure, fulness, tension, or even pain. At the same time there may be belching, nausea, heartburn, vomiting, disturbances of secretion, of the appetite, etc. Secondary or reflex symptoms in other organs may also appear, as headache, palpitation, insomnia, psychic depression, and a variety of other nervous symptoms. All these symptoms coincide with the period of digestion and always follow the introduction of food. At the same time the quantity and the quality of the food do not seem to exercise any marked influence on the severity of the symptoms. We see, therefore, that the condition that we call "nervous dyspepsia" to-day is a combined neurosis of the stomach, in which sensory, motor, and secretory perversions occur at the same time. The subjective sensory symptoms of irritation, however, are always the most conspicuous.

Some authors have objected to retaining Leube's name "nervous dyspepsia" for the form of gastroneurosis that he described, chiefly because sensory disturbances are present alone, and disturbances of the chemism and of the motility of the stomach are absent. This objection, however, becomes invalid as soon as we extend the definition of nervous dyspepsia. I consider this name correct even for Leube's original form, for dyspepsia signifies nothing more than disturbed or difficult digestion, and digestion is certainly disturbed and difficult if the patient suffers pain, belching, or other disagreeable symptoms during digestion. Leube himself has recently extended the definition of nervous dyspepsia to other forms than the one he originally described. Some authors prefer the name "Neurasthenia Gastrica."

Nervous dyspepsia, therefore, is a combined gastric neurosis in which subjective symptoms—namely, sensory disturbances of the stomach—are always present. The latter, however, may be combined with secretory and motor disturbances, as belching, vomiting, peristaltic unrest, and spasm of the pylorus. It is impossible to decide whether or not these symptoms are the result of increased irritability of the sensory nerves. I do not agree with certain authors who claim that motor disturbances do not properly belong to the picture of nervous dyspepsia unless they are the direct result of increased sensibility of the stomach. As a matter of fact, symptoms of motor irritation are frequently observed in nervous dyspepsia; true, they may be absent, but, as a rule, they are present.

Nervous dyspepsia occasionally appears as an independent disease, an apparently independent neurosis. It usually appears together with other symptoms of general nervousness, hysteria, or neurasthenia. It may be due to reflex irritation from other organs, in particular the female sexual organs. In all these cases, however, we are dealing with increased nervous sensibility. Immaterial whether nervous dyspepsia is caused reflexly by some disturbance of the sexual organs or whether it originates from sexual excesses or mental overexertion, we always find that the nerves of the stomach are abnormally irritable. The displacement of the uterus and the condition of neurasthenia do not *per se* produce nervous dyspepsia; this can only occur if the stomach is the *locus* *minoris resistentiæ*, for only then can a physiologic process like normal digestion cause disagreeable or even painful subjective symptoms; only then can irritants that ordinarily cause a normal secretion of gastric juice and normal movements of the organ produce abnormal secretion of gastric juice, spasmodic contractions of the stomach, etc. It is immaterial whether hysteric or neurasthenic symptoms or some sexual disorder are present or not. We frequently see hysteric paralysis of an extremity, or hysteric paresis of the vocal cords, together with other hysteric symptoms, but we may also see these lesions alone. The lesion, therefore, must be interpreted in the same way in both instances. A normal healthy subject will never become afflicted with nervous dyspepsia; a certain nervous predisposition to disorders of this character must always be present. This predisposition may be congenital or may be acquired as the result of exhausting diseases, frequent psychic shocks, mental overexertion, sexual excesses, the abuse of alcohol, tobacco, etc. Severe infectious diseases, like malaria, and chronic diseases, like anemia and chlorosis, may all lead to nervous dyspepsia.

Most authors agree that nervous dyspepsia is a disease that is frequently encountered, but I do not agree with them, for I have frequently seen cases in which the diagnosis "nervous dyspepsia" was made by a number of physicians, but in which finally some organic disease was found after all. It is almost impossible to avoid error in these cases even if they are most carefully examined. In many instances a direct cause for the nervous dyspepsia cannot be discovered. [These cases are more common in America than in Germany.—Ed.]

The disease-picture is very variable, just as the disease-picture of hysteria or neurasthenia. In many cases the patients merely complain of vague, general disagreeable sensations after eating, or a feeling of pressure, of fulness, of tension, or they suffer from belching. In other cases there are nausea, belching, a tendency to vomit, reduced appetite, and a feeling of unrest in the stomach, with occasional attacks of vomiting. All these symptoms, as a rule, appear after eating, sometimes very soon afterward, and persist until the ingesta have left the stomach. In contradistinction, however, to other diseases of the stomach, the quality and the quantity of the food seem to be without influence on the severity of the symptoms. Sometimes the patients feel very much distressed after eating a small quantity of some very digestible article of food, at other times large quantities of indigestible food can be borne without discomfort. If these patients are in good humor, they can eat a most elaborate dinner without distress; at other times, when they are excited, very light food may produce a variety of symptoms. This variability is very characteristic for this form of stomach disease as contrasted with organic diseases of the stomach.

Occasionally the patients suffer distress when the stomach is empty. Boas goes so far as to classify painful sensations that occur when the stomach is empty as a particular form of gastric neurosis. Painful sensations that are experienced particularly when the stomach is empty,—that is, contains no food,—are seen in hypersecretion. Here the acid gastric juice present in the stomach produces pain, probably by causing spasmodic contraction of the pylorus. It is uncertain whether spasms of this kind ever occur when the stomach is absolutely empty; this could only be decided by pumping out the stomach at the time of an attack. Occasionally attacks of pain occur when the stomach contains no food, even though the secretion of gastric juice is found to be normal. Such cases, however, are certainly rare. The suspicion must always be harbored that some organic disease of the stomach, an erosion of the mucosa, etc., exists in these cases.

The individual symptoms in nervous dyspepsia fluctuate in severity, so that it is almost impossible to describe all the different variations that may occur. Pain is the most conspicuous symptom. It may vary in intensity from a feeling of mild distress, or pressure, or fulness in the upper abdominal region, to very violent distress and discomfort, or violent attacks of severe pain. The pain is particularly violent if there is hyperacidity. The distress sometimes begins immediately after eating, sometimes later, and usually terminates as soon as the ingesta are propelled into the intestine. Nothing abnormal can be found in the upper abdominal region on objective examination. We rarely find circumscribed painful pressure-points, but we do occasionally find diffuse painfulness to pressure that extends over the whole region of the stomach but is confined strictly to the boundaries of the organ. Sticker[1] was the first to call attention to the latter symptom in these cases of nervous hyperesthesia (as this disease has also been called). Belching is frequent in nervous dyspepsia, and may be very obstinate in some cases. Pyrosis is less frequent. As a rule, the patients complain of nausea and a tendency to vomit. Vomiting itself is not frequent. Among others, von Leyden has shown that in many cases repeated attacks of vomiting may occur, particularly if there is hyperacidity.

The appetite varies greatly; it may be very good, may be increased, may be reduced, or may be completely absent. As a rule, it varies at short intervals. Occasionally there are attacks of bulimia.

The thirst is sometimes increased, sometimes not. The tongue is usually clean. If it is coated, we must always suspect some complication.

The secretion of gastric juice is, as a rule, normal. In some instances hyperacidity is seen, less frequently subacidity. Whether anacidity is ever combined with complete absence of the ferments in purely nervous disorders of the stomach has not yet been determined, although this is probable.

The motility in the majority of cases is normal, as the stomach is usually found empty seven hours after a test-meal. Even though this is the case, however, we may find hypermotility, and as a matter of fact we frequently do find this condition, particularly in hyperacidity. Atony of the stomach is rare.

As a rule, however, the symptoms are not limited to the stomach in nervous dyspepsia, but involve the intestine. The patients complain of constipation that occasionally alternates with diarrhea. They also complain of a variety of abnormal sensations in the abdomen together with other symptoms, as headache, vertigo, psychic depression, insomnia, lassitude, depression, palpitation, etc.

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[1] *Loc. cit.*

The course of the disease is usually slow. The symptoms vary at different times, and the patient may feel relatively well during one period of time and suffer much distress at another. The disease-picture varies even at the time of greatest distress; to-day we may see one symptom, to-morrow another. The mood of the patient seems to exercise a marked influence on the symptoms, and in jolly company the patient may forget his trouble. These patients are usually very disagreeable company in their homes.

Notwithstanding the numerous complaints of the patients the general health and the general state of nutrition frequently remain almost unimpaired. This may be considered to a certain degree a peculiarity of nervous dyspepia. Of course, if the disease persists very long, and if the appetite is very much reduced, or if the patients suffer from insomnia, the general nutrition will suffer; but these are exceptional cases.

The diagnosis can only be made from the general disease-picture, not from any one symptom nor from the analysis of the stomach-contents, however important this latter procedure may be. Even if the stomach is found to be empty seven hours after a test-meal, this does not by any means demonstrate that the disease is nervous in character nor that digestion is normal; in hyperchlorhydria, as we know, the stomach may be found empty after seven hours or even before. In many cases digestion is normal; in some cases we find hyperacidity, and occasionally subacidity. It may be considered characteristic for the nervous nature of the disease that the secretion of gastric juice and the motility of the stomach seem to vary at short intervals. Another important point is that the symptoms of the patients are more or less independent of the character of the food. The patients may be able to eat indigestible food with impunity on one day, and complain of a variety of local and general symptoms the next day even after eating a light meal. The mood of the patient, his surroundings, and other factors seem to exercise a marked influence on the symptoms. Occasionally a change in the condition of the patient occurs without any apparent cause; he may be enjoying splendid appetite and be sleeping very well, and then suddenly develop a variety of distressing symptoms; or the reverse may occur. The presence of nervous, hysteric, or neurasthenic symptoms I do not consider very important. It is true that symptoms of this kind can usually be discovered in the majority of these cases, but this does not demonstrate by any means that the dyspeptic symptoms are nervous in character, for organic disease of the stomach may be present even in hysterics and neurasthenics.

If the disturbances involve the sensory sphere alone, and if secretion and motility are normal, the diagnosis is relatively easy, for thereby all those diseases of the stomach that are complicated by disturbances of secretion and motility are *a priori* excluded; the diagnosis is more difficult if gastric secretion and motility are perverted, for the disease may then be confounded with ulcer, carcinoma, or chronic gastritis. One of the most important features of the differential diagnosis is the fact that the course of all the last-named diseases is more uniform than that of nervous dyspepsia.

If symptoms of ulcer are pronounced, errors in diagnosis should not occur. But even if circumscribed painful pressure-points, hematemesis, and other typical symptoms are absent, we shall find that in ulcer the attacks of pain are more dependent on the quality of the food than in nervous hyperacidity, and that the pain occurs more regularly and at a definite period of digestion. It is less severe if the diet is liquid and non-irritating, more severe if it is coarse and irritating. Vomiting is more frequent in ulcer than in nervous dyspepsia, and always occurs at approximately the same period of digestion. In ulcer, moreover, the appetite is, as a rule, good and the patients refrain from eating merely because they dread the pain. In ulcer, too, we find the course of the disease favorably modified by proper treatment and diet, whereas this is not the case in nervous dyspepsia. The presence of hyperchlorhydria is of no value in the differential diagnosis, because it may also be present in nervous dyspepsia. If hyperchlorhydria is absent, this may be considered a point in favor of the nervous character of the dyspeptic symptoms, although it does not necessarily exclude ulcer. In the majority of cases the differential diagnosis between ulcer and nervous dyspepsia should be comparatively easy, provided all these points are considered. In many instances the patients will have to be kept under observation for a long time, and the stomach-contents repeatedly examined before a definite decision can be arrived at.

It is not, as a rule, difficult to differentiate nervous dyspepsia from carcinoma; in the earlier stages some difficulty may be encountered, but in advanced carcinoma all the symptoms are typical. If there is vomiting of coffee-ground material, absence of free hydrochloric acid, lactic acid fermentation, we shall have carcinoma in mind even if we cannot feel a tumor. There are, however, many cases of carcinoma in which the first and only symptom is reduced peptic power, or rather absence of free hydrochloric acid. The same may occur, though not so frequently, in nervous dyspepsia. It is immaterial whether or not other nervous or neurasthenic symptoms are present, for a neurasthenic may develop carcinoma. The absence of free hydrochloric acid merely demonstrates that the secretion of gastric juice, or, rather, the production of hydrochloric acid, is reduced. We favor the diagnosis "carcinoma" if the symptoms appear in an old person, and if we are told that the patient was perfectly well until a few months ago, and then developed mild dyspeptic symptoms that gradually increased in severity, and if we find, in addition, that free hydrochloric acid is constantly absent, and that on subsequent examinations the secretions of hydrochloric acid seems to become less and less. If we observe these cases for a long time, we can usually arrive at a positive decision.

The differential diagnosis between nervous dyspepsia and chronic gastritis is not so difficult. In the latter disease some direct injury can usually be discovered that causes gastritis. The vomit or the stomach-contents that is aspirated usually shows reduced hydrochloric acidity, and, as a rule, contains more or less abundant quantities of mucus. The appetite is reduced, the patients can tolerate only very digestible food, and complain of severe distress as soon as they eat anything indigestible. We need not enter into all these details. If all the factors are carefully weighed, and particularly if the stomach-contents is systematically and repeatedly examined, nervous dyspepsia and chronic gastritis should never be confounded.

The prognosis of nervous dyspepsia is favorable as far as longevity is concerned. Personally I have never seen a case that led to death. Some authors, however, as, for instance, Fenwick, have reported fatal cases. The prognosis is unfavorable in regard to the duration of the disease and the liability of recurrences.

Treatment should, in the first place, be directed against the primary cause. In women sexual disorders are usually made responsible for nervous dyspepsia, so that those who harbor this view usually advise treating the local disorders first. This view, however, is only partially correct. Certain diseases of the sexual organs may lead to nervous dyspepsia in one case and not in another. This is due to differences in the individual irritability of the nervous system. Some people are more nervous than others, so that reflex irritation occurs more readily and becomes manifest by disorders in different areas of the body. Local treatment of the sexual disorder may lead to temporary improvement and even a cure of these reflex disorders, but as the nervous predisposition remains there will always be a tendency to recurrence. If the patient is afflicted with some serious disorder of the sexual organs, this may be cured, but a positive and permanent cure of nervous dyspepsia should not be expected from this method, just as little as we can expect to cure hysteria by curing some concurrent sexual disease. All that we can do is to remove the starting-point for reflex nervous disorders; the nervous predisposition always remains. From this point of view we can understand the temporary success of certain operations, and can also understand why recurrences appear nevertheless.

If mental overexertion, business cares, and excitement, [eye-strain], an irregular mode of life, sexual excesses, abuse of alcohol and tobacco, lead to weakness of the nervous system and reduce its tone, the physician must insist on curing all these habits and avoiding all these injuries. In mild cases this is all that is needed for effecting improvement. Those patients in particular who perform much mental labor and carry on a large business, lead an irregular life, or go much into society, will be very much benefited by a sojourn at the seashore or life in the country for a few weeks. In this way the nervous exhaustion is relieved and nervous dyspepsia cured; as soon, however, as these patients return to their old mode of life the old symptoms reappear, and are usually worse than before. In cases of this kind the physician should attempt to change the mode of life altogether and for all time. It is almost impossible to designate how this should be done. If the patients have confidence in their physician, it is a comparatively easy matter. Numerous methods have been devised for supporting and strengthening the nervous system; among them I mention a variety of hydriatic procedures, massage, electric treatment, general faradization. Psychic treatment is still more important than all these methods, for the personal influence of the physician is of fundamental importance in the treatment of these cases. Only if the patient has full confidence in the physician can we expect any good results; if the physician knows this to be the case, he can give his orders with a certain amount of authority, and can feel that his directions will be carried out.

There is no particular diet for these cases. The patients must be taught what to eat and how to nourish themselves. A strengthening diet should always be given, and an irritating diet should be avoided. Occasionally a fattening cure may be indicated, particularly in advanced cases of anorexia (see above). In very severe cases of nervous dyspepsia that are complicated with anorexia, in which the patients are very much reduced, artificial feeding through the sound may be necessary, but this contingency will arise in exceptional cases only. If milk is well borne, it should be given in large quantities wherever nutrition is reduced. Drugs may be called for by certain symptoms, but not by nervous dyspepsia itself. The indications for using different drugs are the same as in other diseases of the stomach that produce definite symptoms; thus, stomachics may be necessary, or if hyperchlorhydria exists, alkaline remedies, etc.

I need hardly mention that the stools should be regulated. Both massage and irrigation may be useful in individual cases. I cannot enter into these details in this place. A course of treatment in some watering-place has often been found advantageous. Iron-waters, sea-bathing, a sojourn in the mountains, a cold-water cure, may all be indicated. The course of treatment should be selected according to the general health of the patient and his strength, and not according to the degree of nervous dyspepsia. It is impossible to formulate any general rules; the only way to treat these cases correctly is to individualize and to weigh carefully all the conditions in each case.

DISTURBANCES OF THE STOMACH-FUNCTIONS IN OTHER DISEASES.

In the preceding sections we have discussed the different diseases of the stomach, and have repeatedly called attention to the relations that exist between the functions of the stomach and general diseases, or diseases of particular organs. We can readily understand why different diseases sooner or later affect the functions of the stomach. It would lead us too far to discuss all the different diseases that exercise a secondary effect on the stomach, and we will consequently limit our discussion to those disturbances and diseases in which perversions of gastric function are particularly conspicuous, and briefly discuss those conditions that have not been considered in previous sections.

The Functions of the Stomach in Febrile Diseases.—In febrile diseases the functions of the digestive organs are nearly always disturbed. The secretion of saliva is, as a rule, diminished, the appetite decreased, and the function of the stomach more or less perverted. The fact that the appetite is reduced in fever does not, of course, justify us in concluding that the functions of the stomach itself are perverted. A healthy subject usually introduces just so much food as is necessary to meet the demands of the organism, so that under normal conditions the appetite is the index of the demand of the body for food. This regulatory function may be perverted in different directions. We have seen that it is altered in the majority of diseases of the stomach. In fever the appetite is also more or less reduced, so that the patients eat less than they require; consequently the organism consumes its own substance and the patients emaciate. It is impossible to say *a priori* whether this reduced demand for food in febrile diseases is due to a perversion of gastric function. It is important to know this both from a theoretic and a practical point of view, for the diet in febrile diseases will depend largely on the secretory and motor powers of the stomach.

The functions of the stomach in fever have been made the subject of particular study within the last decade. Manassein[1] was the first to perform investigations in this direction. His work was all done with animals, and he found that the gastric juice of dogs with fever did not act as powerfully as the gastric juice of healthy animals, and that it required the addition of hydrochloric acid to become as active as normally. Uffelmann[2] studied the same question in human beings with fever, and his results are, of course, more important. In some instances he found a reduction, in others an increase, of the hydrochloric acid. Van der Velden[3] found hydrochloric acid absent in a case of typhoid fever with gastrectasy, although the gastric juice of this patient contained free hydrochloric acid before the onset of the fever. Hydrochloric acid in this case remained absent throughout the course of the febrile process and during the first week after the temperature had become normal.

Sassezki[4] studied 9 febrile cases with varying results. He did not find a reduction of hydrochloric acid in all the cases. In some of them hydrochloric acid was deficient, especially if the patients suffered from dyspepsia.

Edinger[5] also performed some investigations on typhoid fever cases in my clinic, and showed that free hydrochloric acid is present in the stomach even during continuous fever or during transitory high temperature. It is true that in these investigations the acid was not quantitatively determined, but merely the presence of free hydrochloric acid established. Gluzinski[6] did not find uniform conditions. Wolfram, who reported these investigations, that were carried out chiefly in cases suffering from acute infectious diseases (and in two cases of chronic febrile disease), states that in the acute infectious cases hydrochloric acid was absent from the stomach during the entire course of the fever (with the exception of the terminal stage of abdominal typhoid), and that all peptic power was lost. If a certain quantity of hydrochloric acid was added, the digestive power became good, showing that the gastric juice still contained pepsin. As soon as the fever stopped the digestive powers of the gastric juice became normal. In contradistinction to these findings in acute cases, Gluzinski reports normal digestive powers of the gastric juice in *chronic* febrile diseases even during the fever. He expresses the opinion that the perversion of gastric juice in febrile diseases is not so much due to the high temperature as to the character of the infection. The investigations of Edinger and Sassezki, that I have reported above, demonstrate that these findings are not universal. We are probably justified in stating that in acute febrile infectious diseases the production of hydrochloric acid is more frequently reduced than normal, and that the secretion of pepsin is, as a rule, unchanged.

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[1] *Virchow's Arch.*, vol. lv.

[2] "Die Diat in acuten fieberhaften Krankheiten," 1887.—"Beobachtungen an einem gastrotomirten Knaben," *Deutsch. Arch. f. klin. Med.*, vol. xx.

[3] *Berlin. klin. Wochenschr.*, 1877.

[4] *St. Petersburg. med. Wochenschr.*, 1859.

[5] *Deutsch. Arch. f. klin. Med.*, vol. xxix.

[6] *Ibid.*, vol. xlii.

It remains to be decided how this decreased secretion is brought about. The most probable assumption is that the fever itself is responsible. Von Noorden[1] showed that a distinct hydrochloric acid reaction can be obtained in fever cases if condiments or spices, much pepper or salt, are administered with the food, and this speaks in favor of this view.

In chronic cases the results vary still more than in acute febrile diseases. In some instances the values for hydrochloric acid are found normal, in others reduced, and no uniformity or regularity has so far been discovered. In many instances the values for hydrochloric acid have been found normal, and the peptic power good even though the fever was relatively high. Possibly the stomach gradually adapts itself to the fever. In addition to the protracted fever other factors, however, undoubtedly play a rôle; for instance, the general strength of the patient, the character of the disease, etc.

Varying results have even been obtained in the same disease. Schetty,[2] in pulmonary tuberculosis, for instance, found the hydrochloric acid production normal in the morning, and in some cases even increased, whether the patients developed much fever or had no fever at all. He also failed to find any relation between the motor powers of the stomach and the fever, or the intensity or the duration of the disease-process. Rosenthal,[3] on the other hand, found free hydrochloric acid constantly in several cases of phthisis that developed no subjective dyspeptic disturbances but suffered from high fever.

Hildebrandt[4] some time ago performed a number of investigations in my clinic that seem to be more valuable because they were carried out with all necessary precautions and with modern methods. He investigated advanced cases of tuberculosis throughout. The results apparently varied in his series, for in some patients he found hydrochloric acid constantly present, in others constantly absent, and in some cases it was present or absent at different times. On careful investigation, however, a certain regularity could be discovered. The cases in which free hydrochloric acid was present usually had no fever, whereas those in which it was absent suffered from a continuous type of fever. Where hydrochloric acid was absent at one time and present at another, it was found that it was absent in those cases in which the evening temperature was high, and present when it was low. These observations indicate that the rise of temperature, and not the disease-process itself, exercises a distinct influence on the secretion of hydrochloric acid, and this view is corroborated by the observation that a reduction of the fever by antipyrin leads to reappearance of free hydrochloric acid. While we are hardly justified in applying the results of these observations to fever in general or to every febrile disease, they show us that the secretion of gastric juice, like the secretion of other digestive fluids, may be reduced by fever.

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[1] *Lehrbuch der Pathologie des Stoffwechsels*, 1893.

[2] *Deutsch. Arch. f. klin. Med.*, vol. xliv.

[3] *Berlin. klin. Wochenschr.*, 1888, No. 45.

[4] *Deutsch. med. Wochenschr.*, 1889, No. 15.

Different febrile diseases influence this secretion in a different way, and even in individual cases the effect of fever is manifested differently, so that in the one case the disturbances produced are mild, in the other more severe. We may summarize the results of all the investigations we have recorded by saying that fever in general depresses gastrointestinal secretion.

The motor function of the stomach seems to be less involved in fever than the secretory function. I failed to observe any prolongation of the time of digestion in the majority of cases that I examined. Immermann,[1] von Noorden,[2] and others have discovered the same. The powers of absorption, however, as far as we can judge by the potassium iodid method, seem to be impaired in fever (Sticker,[3] Zweifel[4]). Sticker has also shown that absorption is more rapid in fever when the temperature-curve is going down rapidly; when it is rising the time of absorption is usually prolonged.

The Functions of the Stomach in Anemia and Chlorosis.—Anemia and chlorosis must be differentiated. Chlorosis is an independent disease of obscure nature and origin, characterized chiefly by deficient regeneration of the blood from causes that remain unknown. Anemia, on the other hand, is due to some direct injury, profuse loss of blood, hemolysis, or other factors that reduce the quantity of blood. The causes of anemia are known, although they cannot be demonstrated in every case. The primary cause of chlorosis is unknown. Chlorosis, too, leads to anemia, to impoverishment of the blood ; but anemia is not necessarily chlorosis.

I call particular attention to these differences because many investigators have been inclined to apply to chlorosis the results of their experiments on animals that were made anemic, and to argue that the disturbances of gastric function discovered in these animals were identical with those found in chlorosis. This view is not justified, for we do not know whether or not the digestive organs are injured in the same way in both diseases. This question cannot be decided by animal experiments, but only by clinical observation on human subjects.

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[1] *Verhandl. des VII. Cong. f. innere Med.*, 1889, p. 219.

[2] *Lehrbuch der Pathologie des Stoffwechsels*, 1893.

[3] *Berlin. klin. Wochenschr.*, 1885.

[4] *Deutsch. Arch. f. klin. Med.*, 1886, vol. xxxix.

We may expect *a priori* to find that anemia and chlorosis will exercise similar secondary effects on the organs of digestion. Both in anemia and chlorosis do we find numerous digestive perversions. In some cases, however, they are completely absent, in others they are transitory and slight, in still others they are very conspicuous. Why this is the case we do not know. It must never be forgotten, however, that the diagnosis of chlorosis and anemia is not always positive. Let me refer to two diagnostic errors that are frequently committed. Cases of latent ulcer are frequently taken for simple chlorosis, and atrophy of the gastric mucosa is taken for severe anemia, chiefly because no typical stomach-symptoms are observed. The latter cases have, of recent years, been made the subject of particular study. If all cases of anemia and chlorosis are "thrown into the same pot," and we content ourselves with determining statistically how many times the secretion of gastric juice is increased, how many times it is normal or diminished, great variations will naturally be found. But even if we differentiate strictly between anemia and chlorosis we may obtain varying results. If anemia, for instance, is the direct result of a serious hemorrhage in a person who was perfectly healthy before, the secretion of gastric juice will not be perverted in the same sense as in a case in which anemia is combined with atrophy of the gastric mucosa. I merely mention this in order to explain the apparently contradictory results that different authors report.

Subjective gastric disturbances and hyperesthesia, more or less severe pain in the stomach, or even gastralgia, are found both in chlorosis and anemia. These symptoms appear more frequently after eating than when the stomach is empty, and, as a rule, in attacks and at irregular intervals, or occasionally at regular intervals for a long time.

The pain may be due to different causes. It may either be a purely nervous hyperesthesia or it may be due to hyperchlorhydria, causing spasmodic contraction of the stomach. In still other cases there may be hemorrhagic erosions or a latent ulcer. Again, the stomach may be displaced—for instance, vertically; this is particularly frequent in women and young girls. As soon as the stomach becomes distended the pyloric portion is bent and violent attacks of cardialgia may supervene. I need hardly mention that we should not confound this pain with pain in other portions of the intestinal tract, particularly in the transverse colon. As a matter of fact, intestinal colic is very frequently confounded with cardialgia.

The only way in which to determine the primary cause of these symptoms is to examine the patient very carefully, both in regard to the secretion of gastric juice and the position, form, and size of the stomach, and the motor powers of the organ.

Aside from pain we frequently find perversions of the appetite in chlorotic and anemic subjects. Many patients crave acid food, and others, on the contrary, food that can bind acid. The only way in which to determine whether these and similar complaints are merely subjective or whether they are due to some functional perversion is to analyze the stomach-contents with care. But even if organic changes are absent and the functions of the stomach are found normal, we do not know by any means that the symptoms are the direct result of chlorosis. The only way in which to determine the relation between the symptoms and chlorosis is to study each case for a long time. In chlorosis and anemia the nervous system may be involved, of course; so that in many of these instances we are possibly dealing with nervous dyspepsia. The only way, however, to determine the real significance of the symptoms is to examine the patients with scrupulous care.

It is important to study the secretion of gastric juice, and particularly of hydrochloric acid, in all these cases, chiefly because the appetite is reduced, and we must know how to feed these patients.

Many authors have accepted Manassein's[1] statement that the production of hydrochloric acid is reduced in animals with anemia, and have applied his results to man, so that they operate on the basis that the production of hydrochloric acid is reduced both in chlorosis and anemia. They even go so far as to administer hydrochloric acid to chlorotics.

While I attach much importance to animal experiments, I do not believe that we are justified in applying the results obtained from animal experiments to pathologic conditions in man. I am particularly opposed to instituting treatment on the basis of such experiments in cases in which clinical examination of the patient himself can teach us much better what to do. In chlorosis, for instance, the view is generally prevalent that the production of hydrochloric acid is reduced. As a matter of fact, I find that the hydrochloric acidity is frequently very great. I refer to my own investigations and the investigations of my pupils, Grüne[2] and Osswald.[3]

When I first made these statements they were opposed by many. Ritter and Hirsch,[4] for instance, found a reduction of hydrochloric acid in 2 cases of chlorosis and anemia, the reduction being greater in simple anemia than in chlorosis. As against this, Schätzell[5] later reported 30 cases of chlorosis from the same clinic (Leube), in which superacidity was present 22 times.

It would lead us too far to enumerate all the different statements in regard to this matter that have been made by various authors. Some of the investigations were performed with methods that were not universally recognized; in other instances anemia and chlorosis were not differentiated. In nearly all the reports careful statements in regard to the motor function of the stomach, etc., are absent. Among the cases reported many were undoubtedly not genuine chlorosis. I think we should exclude anemia from our studies, as it is not, like chlorosis, a disease *sui generis*, and should include only pure and uncomplicated cases of chlorosis in deciding this question. The significance of anemia we know varies in different cases, so that we may expect *a priori* to find great variations in the secretion of gastric juice.

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[1] *Virchow's Arch.*, vol. lv.

[2] *Inaug. Diss.*, Giessen, 1890.

[3] *Münch. med. Wochenschr.*, 1894.

[4] *Zeitschr. f. klin. Med.*, vol. xiii.

[5] *Inaug. Diss.*, Würzburg, 1892.

It may be considered established that in chlorosis there is no lack of hydrochloric acid.[1] I have found hyperacidity in the majority of cases of pure chlorosis, and cannot say why so many other authors have found such varying results, as they did not publish all the details of their different cases. At all events, hydrochloric acid is not indicated in the treatment of chlorosis. This drug and other remedies intended to stimulate gastric secretion were formerly very popular.

In anemia the findings vary greatly, as is to be expected. Anemia is a sequel of a variety of different diseases, so that in some cases the secretion of hydrochloric acid is normal, in others decreased, and rarely increased. I have found the production of hydrochloric acid most frequently decreased in anemia. The relation between achylia gastrica and anemia is still obscure. I refer for this matter to the section on Achylia Gastrica.

The motor function of the stomach, to judge from my observations, is usually normal or occasionally increased in nearly all cases of chlorosis and in the majority of cases of anemia. Even if the secretion of gastric juice is greatly reduced, or if there is complete achylia gastrica, the motor power may be found normal. We must conclude, therefore, that wherever the motor power is perverted this is not due to perversions of secretion, but to some other complication. In achylia gastrica, for instance, in which the secretion of gastric juice is reduced or almost inhibited, disturbances of motility can, of course, more readily occur than if the secretion of gastric juice is normal or increased.

We may consider it established, therefore, that in simple chlorosis the secretory and motor powers of the stomach are not, as a rule, decreased; on the contrary, more frequently increased. Nevertheless dyspeptic symptoms may develop. The complaints of the patient are out of proportion to the functional disturbances in the stomach. In cases of this kind, therefore, we are justified in giving frequent meals that are not too voluminous. We will often see the symptoms disappear rapidly and the patients recover under this regime, whereas the symptoms persist if the stomach is spared. In other cases of chlorosis we quite frequently find anomalies in the position of the stomach—gastroptosis. Under these circumstances propulsion of the ingesta is rendered more difficult, and this feature must be considered in prescribing the diet. Gastroptosis is a frequent complication of chlorosis.

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[1] Schneider (*Virchow's Arch.*, vol. cxlviii.) found anacidity in 54.2 per cent. of his cases. I fail to understand these results, for I have never found true anacidity in chlorosis.

Dilatation of the stomach, on the other hand, is always due to some particular cause other than chlorosis.

In anemia we find greater variations in regard to the stomach than in chlorosis. It is particularly important, therefore, to study carefully the functions of the stomach, its size, position, and outline in each case of anemia. Only if this is done can we prescribe a rational diet.

The Functions of the Stomach in Heart-lesions.—A long time ago I[1] called attention to the fact that the secretion of gastric juice, or rather the secretion of hydrochloric acid, is reduced during the stage of compensation of certain lesions of the heart. I described 2 cases that demonstrated this finding. Hüfler,[2] guided by his experiments in the clinic at Erlangen, claims that the production of hydrochloric acid is reduced or inhibited in all diseases of the heart, whether or not they involve the valves or the muscles, and that this gastric perversion is due to stasis even in cases in which stasis is not otherwise pronounced.

Hüfler's methods of examination were not, in my opinion, sufficiently accurate to enable him to determine this question. Aspiration of the stomach-contents two hours after a test-meal certainly yields no positive information, so that I hardly feel justified in discussing Hüfler's experiments in detail. Einhorn,[3] Adler and Stern,[4] and others have performed similar experiments.

The results of all these investigations, including my own, vary greatly. In the majority of cases the secretion of gastric juice and the motor powers of the stomach were found normal, particularly if the heart-lesion was well compensated. In later stages, if compensation was disturbed, the secretion of gastric juice was found reduced, particularly if symptoms of stasis were very marked and persisted for a long time. It was often found that the stomach contained free hydrochloric acid after a test-breakfast, but not after a large test-meal. This seems to indicate that the stomach can perform its functions to a certain extent, but that it cannot master large meals—in other words, that its powers are slightly reduced. This observation shows us again that a test-breakfast may aid in deciding certain questions—for instance, whether or not the gastric mucosa still secretes gastric juice—but that it does not enable us to formulate a positive judgment in regard to the normal powers of the stomach. This is one of the chief reasons why both meals should be administered in every case of stomach-trouble.

The Functions of the Stomach in Tuberculosis.—Much has been written in regard to the functions of the stomach in phthisis, and the results published vary greatly. Clinical observation alone teaches us that gastric disturbances may be severe or slight, or altogether absent in phthisis. Frequently phthisis begins with gastric disturbances, or rather gastric symptoms are the most conspicuous symptoms at the onset of the disease. The patients complain of heartburn, belching, frequent nausea, pressure and fulness in the region of the stomach, loss of appetite or fluctuations of the appetite, occasionally even of vomiting. The lung symptoms at the same time are so insignificant that the patients overlook them. This condition has been called pretuberculous dyspepsia. Sée[1] has proposed the name "latent dyspeptic phthisis" for cases in which the phthisic process is masked by dyspeptic symptoms.

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[1] *Zeitschr. f. klin. Med.*, vol. xi.

[2] *Münch. med. Wochenschr.*, 1889.

[3] *Berlin. klin. Wochenschr.*, 1889.

[4] *Ibid.*, 1889.

In the later terminal stages of the disease we also see dyspeptic symptoms. At this stage the appetite may be lost completely; the patients complain of pressure and fulness in the epigastrium, nausea, and vomiting. These symptoms may be nervous in character; or they may be due to the fever, or to distinct gastric changes, as amyloid degeneration of the mucosa. Edinger[2] has reported a case of this kind from my clinic. Or again, atony and ectasy may be present as complications.

Only direct examination can reveal the primary cause of the dyspeptic disturbances. The results of this examination may vary greatly. Brieger[3] has reported a large number of investigations in the functions of the stomach in phthisis. He studied 64 cases and performed 300 individual examinations. Klemperer[4] studied 14 cases.

Of Brieger's 64 cases, 31 were cases of advanced phthisis with continuous fever, 27 of moderate phthisis with more or less intense fever, and 6 incipient cases without fever. Brieger summarizes the results of his investigations as follows:

"Normal chemism was found in only 16 per cent. of the advanced cases, whereas in the others there was more or less insufficiency; in 9.6 per cent. of the cases complete absence of the normal products of gastric secretion was found. In the moderate cases gastric juice was normal in only 33 per cent.; in all others there was perversion of gastric secretion that varied in intensity, and in 6.6 per cent. the normal products of secretion were completely absent. In the initial stages the number of cases with normal secretion was equal to that with perversions."

Of Klemperer's cases, 10 were in the initial stages, only 3 were in advanced stages, and 1 was moderate. In the beginning the secretory powers of the stomach were usually found increased, frequently normal, and rarely reduced; in the terminal stages always greatly reduced.

Klemperer also found the motor power of the stomach reduced in all forms of phthisic dyspepsia. In the initial stages, it is true, this reduction was very slight. True dilatation was absent in all cases. In the terminal stage the motor powers of the stomach were greatly reduced. Brieger also found the motor action greatly retarded in cases in which the secretion of gastric juice was greatly reduced.

My own experience agrees more or less with all the essential points of these investigations. Hildebrandt[5] has shown by his investigations in my clinic that free hydrochloric acid may be present even in pronounced phthisis, but that in cases with continuous fever free hydrochloric acid is usually absent. That the secretion of gastric juice is directly influenced by the fever was shown by the experiment with antipyrin, for here the reduction of the temperature usually, though not always, led to an improvement in the secretion of gastric juice. Gastric secretion may, of course, also be reduced by amyloid degeneration of the mucosa or by gastritis.

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[1] Sée, "Bacillare Lungenphthise," 1886. Translated by Salomon.
[2] *Berlin. klin. Wochenschr.*, 1880; *Deutsch. Arch. f. klin. Med.*, vol. xxix.
[3] *Deutsch. med. Wochenschr.*, 1889, No. 14.
[4] *Berlin. klin. Wochenschr.*, 1889, No. 11.
[5] *Deutsch. med. Wochenschr.*, 1889, No. 15.

We see from all this that the functions of the stomach may be damaged by a number of factors in bacillary phthisis. The primary cause of dyspepsia in each individual case can only be determined by careful examination of the gastric juice. It is important for practical purposes to know this primary cause, for forced alimentation should only be performed if the secretory and motor powers of the stomach are thoroughly understood. It is certainly rational to institute this treatment in cases of phthisis that are much emaciated, and to do everything to raise the nutrition of these patients; but if it is done at all, the powers of the stomach should certainly be carefully studied and considered.

The Functions of the Stomach in Diabetes Mellitus.—Very important work has been done on the relation between diabetes mellitus and the functions of the stomach. Honigmann,[1] Krause[2] in my clinic, Rosenheim,[3] Gans,[4] Sée,[5] Cantani,[6] have all worked along these lines. They were particularly interested in this subject, because polyphagia is so frequent in diabetes, and it seemed important to investigate the functions of the stomach in this disease. *A priori* an increase of functional activity was to be expected more than a decrease. It appears, however, that the investigations of different authors have yielded varying results. I,[7] for instance, found pronounced hyperacidity in a diabetic case at the time of increased appetite. As soon as the appetite lessened, normal acidity was reëstablished. Rosenstein examined 10 diabetics, and found the secretion of gastric juice normal in 4 and abnormal in 6. In 2 of these cases free hydrochloric acid was absent for a long time, but appeared again later on. In a third case free hydrochloric acid was sometimes present, sometimes absent. Rosenstein attributes this changeability to a neurosis of the stomach analogous to other diabetic neuroses. In a fourth case free hydrochloric acid was only found toward the end of the investigation. Here postmortem examination revealed a localized atrophy of the gastric glands, but the lesion was too small to account for the deficient secretion. In 2 other cases Rosenstein failed to find free hydrochloric acid at any time. The autopsy findings in 3 other diabetics are also important, although the gastric juice was never examined during life, for in these 3 cases advanced interstitial gastritis was found involving large areas of the mucosa; in these areas the glands were destroyed.

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[1] *Deutsch. med. Wochenschr.*, 1890, No. 43.
[2] *Inaug. Diss.*, Giessen, 1890.
[3] *Berlin. klin. Wochenschr.*, 1890, No. 13.
[4] *Verhandl. d. IX. Cong. f. innere Med.*, Wiesbaden, 1890.
[5] Sée, "Les maladies de l'estomac, jugees par un nouveau réactif chimique." Communication to the Académie de Médecine, January, 1888.
[6] *Il Morgagni*, 1883.
[7] *Zeitschr. f. klin. Med.*, vol. xii.

Rosenstein, therefore, believes that the gastric symptoms in diabetes originate in different ways. If free hydrochloric acid is always absent in diabetes, we must think of atrophy of the glandular apparatus of the stomach due to interstitial inflammation. In other cases in which free hydrochloric acid is absent for short or long periods of time, this must be considered to be due to a neurosis of the stomach.

Gans examined 10 cases of diabetes of different degree. The results of this investigator are more or less uniform, for he found that the composition of the gastric juice was in no wise related to the production of sugar, and that fluctuations in the chemism of the gastric juice occurred in the same individual without any relation to the percentage of sugar excretion. In 6 cases distinct hydrochloric acid reactions could be obtained; in 4 cases the reactions were negative. The motor power of the stomach was normal in all of the cases.

Honigmann examined 8 cases of diabetes, some mild, some of medium severity. He performed 45 single analyses of the stomach in all. Four of these cases were normal as regards gastric secretion; only here and there was slightly increased hydrochloric acidity found. In 3 of the cases free hydrochloric acid reactions were never obtained, but the motility of the stomach was found to be good. In the remaining case the gastric juice was found perfectly normal the first time it was examined, but free hydrochloric acid was absent in the 11 examinations that were made afterward; the total acidity at the same time was very slight.

We are unable to draw any general conclusions from these investigations. We may, however, summarize the results as follows:

In diabetes no direct connection can be shown to exist between polyphagia and the amount of hydrochloric acid secreted. A definite relation was found only in the first of the cases that I reported. While it is true that the acidity of the gastric juice is occasionally increased, this can hardly be considered the cause of polyphagia; it is more probable that the stomach, under these circumstances, reacts to the increased work it has to do by a secondary increased secretion of gastric juice.

Honigmann has argued that we are not justified in diagnosing atrophy of the gastric mucosa even in the cases of anacidity in which the color-reactions are negative. Only if no hydrochloric acid whatever is produced should we make such a diagnosis. Many of the investigations that have been published so far are worthless in this respect, although a few of them seem to indicate that atrophy of the gastric mucosa may occasionally occur in diabetes. Future investigation will have to show whether or not in many cases the absence of free hydrochloric acid is due to the increased motility and the rapid propulsion of the ingesta. This is Honigmann's view, for he ascribes the lack of hydrochloric acid, or rather the absence of a free hydrochloric acid reaction, to this increased motility. Whether or not Rosenstein is correct when he calls the cases in which the analysis of the gastric juice reveals great variations, neuroses of the stomach, remains to be determined. Honigmann warns particularly against drawing any general conclusions of this character, as errors in method are so liable to occur.

The fact that the motor power of the stomach is so good is remarkable. This was observed to be the case both in our patients and in the cases reported by Gans. It explains why the stomach, despite the existence of polyphagia, can digest such large amounts of food, and why digestion remains undisturbed even though the secretory powers of the stomach are reduced.

The Functions of the Stomach in Diseases of the Kidneys.—Very little work has been done on the relationship between nephritis and perversions of gastric function, although gastric disturbances are relatively frequent in nephritis. This subject is particularly important because the diet has to be so carefully considered in treating kidney cases. Sée[1] and Lenhartz[2] each examined 1 case and found anacidity. Schneider[3] found hyperacidity in 1 case and anacidity in 3 cases. Krawkow,[4] Zipkin,[5] and in particular Biernacki,[6] have performed more careful and exhaustive investigations. Zipkin found hypoacidity once in 4 cases, and normal or increased hydrochloric acid secretion in all the rest. Krawkow found normal acidity 4 times in 26 patients, hyperacidity 14 times, and anacidity 8 times. Biernacki has performed the most exhaustive investigations. He studied 25 cases of nephritis; among them cases of acute and interstitial nephritis. He found that the secretion of gastric juice is in general reduced in all cases of inflammation of the kidney, and that the degree of this reduction varies in different cases. Occasionally hydrochloric acid was found absent. He also showed that the quantity of free hydrochloric acid seemed to be reduced in proportion to the extent of edema, the excretion of albumin, and the reduction in the total quantity of urine excreted.

Biernacki also found that free hydrochloric acid is usually absent in severe cases of nephritis during the stage of edema, and is present in the milder cases in large or small quantities. The secretion of pepsin is also reduced even in mild cases. The motor power of the stomach, on the other hand, is frequently found increased, not only in mild cases, but also in old, chronic cases. Biernacki expresses the opinion that the reduction of gastric secretion during the period of oliguria is largely due to the depression of glandular function by toxic metabolic products. He also believes, however, that the reduction in the secretion of gastric juice may be due to organic changes of the stomach in those cases in which nephritis persists for a long time, in which the blood-changes are considerable, and in which, consequently, the nutrition of all the tissues suffers.

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[1] Sée "Les maladies de l'estomac, jugées par un nouveau réactif chimique." Communication to the Académie de Médecine, January, 1888.

[2] *Deutsch. med. Wochenschr.*, 1890, and *Schmidt's Jahrbuch*, 1890.

[3] *Virchow's Archiv.*, vol. cxviii.

[4] *Inaug. Diss.*, St. Petersburg, 1891.

[5] *Inaug. Diss.*, Würzburg, 1894.

[6] *Centralbl. f. klin. Med.*, 1890; *Berlin. klin. Wochenschr.*, 1891.

We see, therefore, that the findings of different authors vary greatly. Whereas Krawkow found anacidity 8 times in his 26 cases, Biernacki found reduction of the hydrochloric acidity throughout. My own investigations yield results that are still different, for I have frequently found normal hydrochloric acidity even in old, chronic cases of nephritis, and in a few, of course, a reduction of the hydrochloric acidity. To judge from my personal experience, deficiency of hydrochloric acid is by no means a regular finding in nephritis.

I do not think it worth while to attempt an explanation of these findings. In the first place, it will be necessary to determine more carefully when and under what conditions the secretion of gastric juice is reduced in diseases of the kidney. The results of all the investigations, moreover, that have been published so far are too contradictory and too scanty to allow us to draw any conclusions in regard to the conditions that favor the occurrence of functional disturbances of the stomach in this disease.

It would lead us too far to discuss all the other diseases that may exercise an influence on the function of the stomach under certain conditions. I have already discussed the influence of diseases of the liver (which need especial attention) on gastric functions when discussing the different diseases of the stomach.

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